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The Medical Clinics of Chicago

JANUARY, 1917

VOLUME 2—NUMBER 4

411881
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PHILADELPHIA AND LONDON

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PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR), BY W. B. SAUNDERS COMPANY, WEST WASHINGTON SQUARE, PHILADELPHIA.

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The
Medical Clinics of Chicago

VOLUME 2 NUMBER 4

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

SPLANCHNOPTOSIS

Varieties and Symptomatology. Physiologic and Anatomic Factors. Relationship of Subjective Symptoms to Objective Findings. "Nervous Dyspepsia." The Floating Kidney. Treatment of Splanchnoptosis in Detail.

October 25, 1916.

I HAVE 3 cases to show you today which represent substantially the same condition, each of them, however, presenting sufficient differences to warrant my showing them to you. All 3 of these cases are women, all private patients, and all have been under observation for at least two or three years. I am presenting them to you with the principal idea in view of illustrating the results of what I regard as the proper management of such cases.

The first patient, Mrs. T., a resident of Michigan, is married, aged forty-two years at the time she came under my observation, which was May 1, 1912. The salient points in her history are as follows:

Family History.—Her father was living and well; mother dead, cause unknown; no sisters or brothers.

Past History.—Her past history was entirely negative except that she had had the ordinary diseases of childhood. She always considered herself as perfectly well up to the time of the birth of her first child.

Present History.—The present trouble dates back many years, having begun shortly after the birth of her first child. After this first delivery, the child being of about normal weight, she

noticed that she "had lost her figure," and about this time commenced to have "indigestion." This indigestion did not seem to be closely associated with her meals, nor was there anything in particular which brought it on so far as she knew. She simply noticed that she commenced to have attacks of difficult digestion, these attacks becoming more and more frequent. She was not very clear as to just how frequent they were at that time, but was quite certain that they were not nearly so frequent or so constant as they were during the year preceding the time that she came under my care.

After the birth of her second child the trouble became worse and she complained of much pain in the back. This pain was aggravated by exercise, jolting, and by riding, especially over rough roads. Ordinary street-car riding increased the pain in the back and the right side. The attacks of indigestion now lost their periodic character and became almost constant. During all this time she was steadily losing weight. She had been an extraordinary healthy young girl, of large frame and substantially built figure. She says she weighed 120 or 125 pounds at the time of her marriage, but since the onset of her trouble had been going down steadily. She thought she weighed about 100 pounds at the time of the birth of her second child. With the birth of her third child, a couple of years later, her symptoms became much worse. The "dyspepsia" became more marked and was practically constant. She complained of a great deal of belching, and her constipation, which up to this time had not been of more than moderate degree, became extreme, so that she had great difficulty in obtaining normal evacuations of the bowels.

She had repeatedly consulted physicians, who prescribed various remedies for her hyperacidity and constipation. Some time after the birth of her third child, approximately four years before she came under my care, she commenced to notice that she was very tender in the right inguinal region, and appendicitis was suspected. This tenderness had the peculiarity of being much more marked when she had been on her feet all day and much better when she got up in the morning. For the few months preceding the time when I first saw her this tenderness in the right

iliac region became more marked, causing the patient to stoop over slightly. She noticed that whenever her abdomen was examined by a physician it hurt her greatly. There was never any vomiting, but nausea was tolerably common. Her menstrual periods were regular, as a rule attended with only a moderate amount of pain, but at times were very painful. There seemed to be no connection between her dyspepsia, the pain in her right side, and the difficult menstruation. Her appetite was very poor, and for the preceding year especially she had been afraid to eat because of the indigestion. All of these causes produced a steady loss of weight, and when she came under my care she weighed $87\frac{3}{4}$ pounds, a loss of between 30 and 40 pounds as compared with her weight when married. A brief synopsis of the results of my examination is as follows:

Examination.—The patient was a rather large and substantially built woman with good bony framework, greatly emaciated. The *face*, *head*, and *neck* presented no abnormalities except that the skin was pale and unhealthy looking. The *chest* was of normal configuration. The *lungs* were entirely negative, the boundaries in their normal position and freely movable. The Krönig fields at the apices were normal, and no evidence of tuberculosis could be found. The *heart* was entirely negative. An *x*-ray examination of the thorax showed no abnormalities.

The *abdomen* was most characteristic. Between the ensiform and the umbilicus it was greatly sunken, below the umbilicus, very protuberant. The flanks, particularly in the region of the kidney, were sunken in, and the anteroposterior diameter at this point greatly diminished. The recti showed a slight diastasis, and on placing the patient in the recumbent position the anterior abdominal wall was found to be in an extraordinary state of flaccidity and relaxation. On demanding of the patient that she keep her knees stiff and raise the legs, it was found that the abdominal muscles were too weak to accomplish this. On palpation, the entire right side of the abdomen, particularly the region about the level of the navel, was tender, and a mass could be felt which was freely movable in all directions and would slip up under the costal arch with great readiness. This mass was

slippery, easy to push down and feel in its entire extent, exquisitely tender, and was of the general shape of the kidney. In size, however, it was fully one-third again as large as the normal kidney.

Inflation of the colon showed this mass to be retroperitoneal, and the ease with which it slipped back under the costal arch left no doubt that it was, in reality, a tumor of the kidney itself. The liver could be felt just below the costal arch. The spleen was not palpable. The gall-bladder region was negative.

At this point it might be well to say that she had had on several occasions small amounts of blood in the urine, not enough to be noticeable to the naked eye, but in quite considerable amounts microscopically.

Let us pause here to consider what this tumor might be. It was really because of this and because of the tenderness that she came under my care. The hematuria, loss in weight, the presence of a tumor, and the enlargement of the liver had led to the suspicion of a hypernephroma. Examination of the mass on the right side showed it to be perfectly smooth; no nodules of any kind could be demonstrated. On the left side the kidney could also be felt for about one-half to two-thirds its extent. The hematuria was distinctly intermittent. It would be present in a single specimen after she had been up and around, and the following morning would disappear. This, of course, spoke strongly against a malignant neoplasm, since the majority of malignant neoplasms, when they once start to bleed, bleed constantly and very materially. There were no attacks of pain of such nature as to lead one to suspect stone with secondary hydronephrosis, and an *x*-ray of the abdomen failed to show the kidney at all. Aside from the small amount of blood occasionally present, the urine was normal—no albumin, no sugar. There was no increase in the blood-pressure, so there was nothing to warrant the diagnosis of a polycystic kidney.

Now, when we consider that we had a patient with a long-drawn-out history of vague "dyspepsia," and loss of weight amounting to 30 to 40 forty pounds over a period of many years, the condition becoming worse with each successive childbirth,

and we find a large, swollen, and tender kidney, the picture is a very characteristic one. This is the picture of displaced kidney which often occurs in splanchnoptosis. The swelling of this kidney was entirely due to venous congestion, possibly aided at times by a slight retention of urine as the result of kinking of the ureter, although I am somewhat skeptical on this latter point. We shall see that the condition is precisely analogous to the enormous enlargement of the liver which occurs in broken-down heart conditions, except that in the case of the kidney the congestion is not due to a broken-down heart, but to interference with the return flow of blood through the renal veins as the result of its displaced condition. I assured this woman's physician that I felt quite confident there was no malignant neoplasm, and that this large and tender kidney was merely the result of a general splanchnoptosis, and we then proceeded to investigate the position of the other organs.

On inflation of the stomach with a double bulb it was found to be far below the navel. Inflation of the colon was unsatisfactory, as we had great difficulty in getting air past the splenic flexure. The *x*-ray plates of the colon after a bismuth meal showed the transverse colon to be V shaped and greatly prolapsed, as could be readily seen.

Further examination of the patient showed nothing special. The blood-count was approximately 4,500,00 red cells, with 11,000 leukocytes and 11.4 grams of hemoglobin per 100 c.c. of blood. There was no abnormal temperature. The stools were negative in every way, especially as to blood. The test-breakfast showed nothing abnormal except a moderate degree of hyperacidity, and in particular it should be noted that the stomach emptied itself in approximately the normal time.

Now, gentlemen, I present this case to you as a typical one of pretty high-grade general splanchnoptosis, and I am showing you the case today to illustrate how I am accustomed to manage such cases and to show you the results achieved in them. Let us leave this patient for a moment and make a little excursion into splanchnoptosis in general, particularly as to its etiology, symptomatology, and treatment. In a general way we may recognize

two great groups: (1) Those in which the condition is congenital and due to a defective body form; (2) the acquired cases. The first group of cases represent in a sense congenital malformations, and their treatment is entirely different from the treatment of acquired cases, and it is to the latter group that I wish to confine my remarks today. In general medical practice the great major-

Fig. 110.—Stomach from Case 1. Patient in erect position.

ity belong to the acquired group. The patient I have just shown you was a perfectly typical and characteristic case. A well-developed, strong, sturdy girl, brought up in the atmosphere of a small town, living a healthy and sensible life, is married, becomes pregnant, and in the course of time is delivered of a normal child. She noticed, as you will remember, that she "lost her figure" at this time. In other words, the entire abdominal wall became

relaxed, so that the upper segment became flattened and the lower segment became protuberant. When we reflect for a moment as to the causes of splanchnoptosis of the ordinary type the following considerations are apparent: If we regard the abdomen as a flattened cylinder we shall see that in the back the

Fig. 111.—Colon: Patient with high-grade splanchnoptosis (typical V shape). Marked ptosis of cecum, transverse colon, and both flexures.

spinal column, the ribs, the pelvis, and the powerful muscles passing from the pelvis to the lower ribs form a strong barrier which cannot yield, and on the sides of the abdomen the ribs come down low, and fairly strong muscles connect the ribs with the crest of the ilium. In front, however, we have entirely different conditions. Not only do the ribs not come down low, but

they bend upward sharply to meet at the sternum, so that there is an enormously greater difference in the distance from the pelvis to the thorax in front than there is at the sides. This is, of course, a necessity, to allow for the expansion and contraction of the abdomen in physiologic states, particularly in pregnancy. This shows at once very clearly the importance of the integrity

Fig. 112.—Typical V-shaped colon in a moderately high-grade splanchnoptosis.

of the entire abdominal wall in maintaining the normal contour of the abdomen. In a nutshell, we may regard the abdomen as rigid in the back and on the sides and closed with a large sheet of relatively thin musculature over the very extensive anterior surface. Anything which interferes with the integrity of this great muscular sheet and allows it to sag removes the support from the

viscera by failing to maintain the pressure which it should normally exert upon them, and the result is that all of the organs are permitted to hang down as far as may be allowed by their other supporting structures.

Let us now consider these other supporting structures. One sees a great deal of discussion in surgical and anatomic literature of the so-called "ligaments" of the various organs, but when we reflect that for the most part these ligaments are nothing but thin folds of peritoneum, with perhaps a certain amount of fat included, we shall see that this name is a highly misleading one, for one is led to think of the ligaments in the sense of the ligaments connecting the joints. Nothing is farther from the fact. The actual supporting power of the so-called ligaments of the various abdominal viscera is not great, and in my judgment a great deal too much stress has been laid upon them in this connection.

Again, we have another factor in the support of the various viscera, particularly the kidneys, which is commonly lost sight of. I refer to the intra-abdominal fat, which in the case of the kidneys amounts to a veritable fat capsule. The perirenal fat is a very definite factor in the support of the kidneys, as is abundantly shown by clinical evidence. Not only this, but the intra-abdominal fat acts as a pad, filling out the interstices between the various viscera in such a way that the anterior abdominal wall can support these latter in the most effectual manner. In my judgment, the two great factors in the production of acquired splanchnoptosis are: first, a flaccid abdominal wall, generally brought about by repeated pregnancies and often associated with weak muscular tone due to anemia, indolent habits of living, etc.; and, second, a great loss in the intra-abdominal fat. The condition is one which comes about gradually, not suddenly, and the patient I have just shown you is a beautiful illustration of this fact. The stretching of the abdominal muscles due to her first pregnancy initiated her dyspeptic disturbances. Instead of taking measures for the correction of this muscular weakness, she strove to hide it by corsets, which, of course, further interfered with the normal functions of these muscles, and in the long run made matters steadily worse.

Symptomatology.—I think it is proper at this stage to discuss the relationship of the subjective symptoms in splanchnoptosis to the objective findings. This is a matter which, since the days of Glenard, has excited much discussion and about which the last word has not yet been said. One group of men regard the nervous symptoms which are so common in these cases as being due to the splanchnoptosis. Other men regard them both as part of

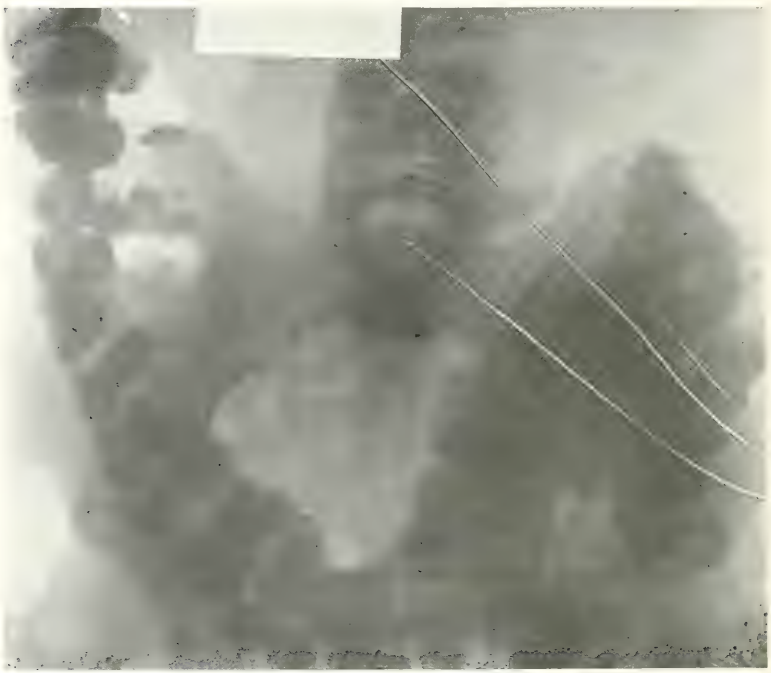

Fig. 113.—High-grade coloptosis. Part of a general splanchnoptosis.

a nervous constitution. My own feeling in the matter is this: Given a strong, healthy individual to begin with, and a clean-cut history of the gradual development of a marked splanchnoptosis, it seems to me that we cannot avoid the conclusion that the dyspeptic disturbances, constipation, and general nervous condition are actually due to the splanchnoptosis. It should not be lost sight of, however, that it is probably not the splanchnoptosis operating in a mechanical sense, but rather its interference with

the normal physiologic processes of digestion and assimilation. I mean by this that the splanchnoptosis when of sufficient degree to produce symptoms at all produces a discomfort in the abdomen which leads the patient to give up one article of diet after another, and this in turn leads to progressive loss in weight and strength, which is probably the most important factor in the production of the nervous symptoms. In a sense we have a vicious circle. Splanchnoptosis creates certain dyspeptic disturbances; these, if neglected, lead to interference with the appetite and possibly interference with assimilation, and the result of this is a steady loss of weight and strength, which may go on to a point which renders the patient's life a burden. That was about the situation with this patient.

It is rare in my experience to have a kidney prolapsed to such a degree as to produce even a microscopic hematuria, and yet there can be no question that such was the case with this woman. Considering the symptoms of splanchnoptosis in general, the first point to be noted is that many cases of moderate degree produce no subjective symptoms whatever. One constantly sees women who have somewhat relaxed abdominal walls in whom the kidney is palpable but not tender, and with the stomach and colon situated a little lower than the average, but showing no symptoms in any way referable to the abnormal position of the viscera. I regard such a condition as scarcely outside the normal limits of the physiologic, and such patients demand no treatment, although if there is a possibility of more pregnancies a course of exercises such as I shall describe to you later is a useful prophylactic measure.

What symptoms can be legitimately attributed to the splanchnoptosis? First and foremost I should say a swollen and tender kidney. I think much too little attention is paid to this. Many instances of backache, with pain and tenderness in the sight side of the abdomen, can be shown to be due to an abnormal kidney, the congestion being due to its abnormal position. The justice of this is readily seen by the fact that if a patient is in bed for a few days the swelling and tenderness disappears, as do also the backache and pain in the right hypogastric and inguinal regions.

Such was the case with this patient. After a week's stay in bed, the swelling getting less every day, the tenderness disappeared entirely, and has remained away up to the present time. The dyspeptic symptoms are characteristic of splanchnoptosis. It is difficult to describe them accurately because they are of a vague nature and are best classed in that motley group of disturbances which we refer to as "nervous dyspepsia." In many instances it is difficult to determine whether the mental condition or the splanchnoptosis is the cause of the dyspeptic condition. It is commonly stated that if the wearing of a belt or properly fitted corset gives relief this may be regarded as proof that the condition was due to the splanchnoptosis *per se*. I find it difficult to procure such relief, so I am in the habit of saying that if a few days' rest in bed upon the same diet which the patient was accustomed to take gives relief, this fact is additional evidence of the dyspeptic symptoms having been due to the altered position of the stomach and other viscera. The shape of the stomach is materially altered in splanchnoptosis, as has been recognized for many years. I can best describe it to you by saying that the fundus is more or less obliterated. It is flattened out, and the greater curvature hangs down so that the general outline of the stomach is U shaped. It is difficult to draw a line between what is strictly normal and what is abnormal. The *x*-ray has taught us to be a little more careful in interpreting moderate displacements of the stomach. Certainly one should not refer to a stomach as displaced until the greater curvature is well below the level of the umbilicus. The colon is affected by the splanchnoptosis in the sense that the transverse colon sags, and when the degree reached is considerable it takes on a V shape, the apex of the V being in the midline between the navel and the symphysis, and the prongs of the V being at the hepatic and splenic flexures. Apparently the colon is greatly lengthened. This is not, however, actually the case. What really happens is that the colon is to a great extent shelled out of the hepatic and splenic fossæ where it normally lies, and instead of curves in these positions we have sharp, angular flexures. This is one of the reasons for the constipation which is so very common in these cases. I have seen

one case in which feces accumulated in such a degree at the apex of the V in the transverse colon as to form a definite tumor mass just at the symphysis pubis, large enough to cause pressure upon the bladder and excite the suspicion of carcinoma of that organ. Indeed, a diagnosis of carcinoma of the bladder was actually made, and it was only when I discovered the great splanchnoptosis that the condition was shown to be due to fecal impaction at this point. The liver and spleen are relatively seldom displaced in acquired splanchnoptosis, and I am not aware of any symptoms which may be properly attributed to such displacements when they do occur.

Possibly the most important symptoms of all are those which go with the impaired nutrition. The condition of splanchnoptosis lasting over a series of years will produce a steady loss of weight with a corresponding muscular weakness and generally impaired nutrition, and this, in many cases, is actually the thing for which the patient seeks relief. I have had many patients come to me with a tentative diagnosis of tuberculosis, and at least one who was suspected of having a carcinoma because of the great loss of weight. If one remembers, however, that this loss of weight does not take place suddenly, or in a year or two, but in the vast majority of cases extends over many years, it will not be difficult to avoid these pitfalls in diagnosis.

Diagnosis.—This brings us to the discussion of the diagnosis of splanchnoptosis in general. If one is familiar with the condition the diagnosis is generally made with great readiness. The examination should be made after the entire intestinal tract has been thoroughly emptied. With the patient on the back, the legs drawn up, and the abdominal muscles relaxed to the utmost, it is an easy matter to palpate the kidney and determine whether it is (1) simply palpable, (2) actually movable, or (3) whether it is so movable as to warrant the name of "floating kidney." This latter, it may be remarked in passing, is rare. There are several methods of examination for the position of the stomach. It may be inflated by the well-known bicarbonate and tartaric acid method or, better still, by the passage of a tube and inflation with a double bulb. Where expense is not an item

the *x*-ray is the most satisfactory method of examination, and it has the additional merit of giving one a permanent record. The position of the bowel can be determined by inflation with air or by filling with water, but neither of these is so satisfactory as the *x*-ray examination after a bismuth meal or bismuth enema. The gastrodiaphane, originally recommended by Einhorn, is fairly satisfactory for a quick orientation of the position of the stomach if one is thoroughly familiar with its shortcomings and the interpretation of the results. I use it less frequently now that the *x*-ray is so readily accessible.

You will see that the real difficulty in cases of splanchnoptosis is not the diagnosis of the splanchnoptosis *per se*, but to decide in how far the symptoms can be legitimately attributed to the displaced viscera. It goes without saying that a painstaking examination of the entire body must be made to be certain that the loss of weight and dyspeptic symptoms are not due to tuberculosis or incipient carcinoma or some allied condition. Having made the diagnosis, the questions of prognosis and treatment present themselves.

Prognosis.—The question of prognosis is, broadly speaking, the question of your ability to carry out what I regard to be the proper management of these cases. In the ordinary treatment, such as is carried out in dispensaries, my experience is that the prognosis is poor as regards very great improvement. Occasionally one can bring about an improvement which is worth while. Given a patient who is intelligent and who can carry out your instructions, the prognosis is excellent. Indeed, I should like to emphasize that I know of no more satisfactory type of case to treat than these. To begin with, the diagnosis has generally not been made with any degree of accuracy. The patient has probably tried many different things, generally unavailingly, and when she commences to improve is usually only too willing to continue the treatment which you have instituted.

Treatment.—The indications for treatment in splanchnoptosis are: first, absolute rest in bed; second, systematic overfeeding in such a way as to throw the least possible strain upon the digestive

organs, and, in particular, to run the least possible risk of dilatation of the stomach; third, systematic exercises designed to strengthen the anterior abdominal wall and thus correct one of the main causal factors of the condition.

In regard to the first indication, the rest in bed, I regard it as essential, inasmuch as it eliminates the effect of gravitation upon the organs and generally in and of itself produces a marked improvement upon the symptoms. I insist upon long-continued rest in bed under proper hospital conditions. I do not allow these patients to sit up, as, of course, sitting up is, so far as the effects of gravitation on the viscera are concerned, identical with standing.

In regard to the second condition, I treat my patients along the same lines that one employs in the so-called "mast-kur." I do not think it is a matter of great importance as to the precise food given, provided that it be given in small amounts and at short intervals. A milk-and-cream mixture is thoroughly satisfactory to begin with, and I generally give it, beginning with a 3- or 4-ounce mixture every hour from the time the patient wakes up in the morning until bedtime at night. A certain amount of individualization is, of course, always necessary. Perhaps the most satisfactory way of stating the treatment would be to tell you exactly what we did in this case. I began with a mixture of milk and cream in the proportion of 2 parts of milk and 1 part of cream, giving an 8-ounce mixture every three hours. Midway between these feedings I gave in the morning a dish of cereal with cream and sugar; at noon a baked potato with plenty of butter and a slice of toast, and this was repeated in the evening. Without going into the precise details of the feeding, I will say that soft foods were gradually added, the proportion of cream was increased until equal parts of cream and milk were taken, the quantities being guided entirely by the gain in weight. I generally expect my patient to gain 1 or $1\frac{1}{2}$ pounds the first week and at least 2 pounds each successive week. This patient, as I stated to you, weighed $87\frac{3}{4}$ pounds on entering the hospital, which was on May 4, 1912; on July 20th she weighed 112 pounds, a gain of approximately 25 pounds. She left the hospital on the

31st of July, weighing about 117 pounds, a gain averaging approximately 2½ pounds weekly, 29 pounds in all.

In the four years which have elapsed since her treatment she has gained weight slowly, and now weighs about 130 pounds. She feels well in every particular, her kidney is no longer palpable, there is absolutely no pain in the lumbar or hypogastric region on the right side, her dyspeptic symptoms are entirely relieved, and she jokingly says that she has grown so heavy that her next move will be to come to me for a reduction cure. As a matter of fact, she now weighs about what she should for her height, age, and general build.

The exercises which I prescribed for her were of a very simple nature, and designed to strengthen not only the recti abdominis but the obliqui and transversalis. I began with having her lie upon a blanket upon the floor (not on a bed or couch, because the body sinks into these too far), and with the knee straight, raise each leg alternately. This is continued for perhaps ten minutes three times a day until the patient's strength increases materially. Then both legs are raised in the same fashion. A little later I added another exercise designed to strengthen the lateral muscles of the abdominal wall. With the patient lying flat on the back, the arms extended at right angles and the palms down, the legs, with knees stiff, are brought up to a right angle and then swung over in each direction as far as possible without rotating the trunk. This is done first to one side and then to the other. Another exercise added later is to flex the legs upon the thighs and the thighs upon the pelvis, at the same time raising the head, as if the patient were endeavoring to touch the ears with the knees. This we call the "ear tickler." Later, as the patient gained strength, I had her crawl or, rather, walk on all fours. The virtue of this is that a person instinctively holds up the abdomen, even without being told to do so, and, in addition, it is a splendid general exercise for patients in this condition. As the patients get a little stronger they can place their feet under a radiator, or have an attendant hold the feet down while lying supine on the back, and then sit up gradually. This is another means of exercising the recti in particular. This requires considerably more strength than the patient

usually has to begin with, and the exercise may be made easier by extending the arms parallel with the trunk above the head, the hands clenched, and swinging the arms up just in advance of the trunk. Patients can frequently accomplish this readily, whereas sitting up with the arms folded across the chest is impossible for them.

These are some of the exercises I employ, and there are, of course, many others which can easily be found in books on kinesitherapy.

The second patient I wish to show you is in many respects a duplicate of the first, but presents certain features which make her case particularly instructive. She was thirty-eight years of age when she came under my care October 1, 1913. Her history was entirely uneventful until after her marriage. She is the mother of two children, and her difficulties commenced after the birth of her second child, about five years before I first saw her. She began to feel very nervous and irritable. She stated that her appetite was fair, but in spite of this she steadily lost weight. As you see, she is rather above the average size, exceedingly well developed and with large bones. Her normal weight was about 155 up to the birth of her second child. After this she developed a nervous and irritable condition which lasted about a year, when she commenced to develop vague dyspeptic disturbances, complained of "gas on the stomach," and a good deal of gas in the intestines. She never had any actual pain, but had a great deal of backache and soreness, with tenderness on the right side of the abdomen, sometimes low down, sometimes higher up. Her menstrual periods presented nothing abnormal. Ordinarily her bowels had acted regularly, but for the previous year or two she had been becoming increasingly constipated.

About two years before I first saw her the discomfort in the right side of the abdomen became so marked that she thought she had some surgical condition and consulted a surgeon. She told me his diagnosis was "floating kidney," and that she was told that the kidney was down below the level of the navel at that time. He advised operation to stitch up the kidney, which was agreed to, and the operation was done a few days later. While in

bed her symptoms were better, but as soon as she got up they returned, and since that time had been getting steadily worse, so that in the year previous to consulting me she had lost about 20 pounds, her weight being then 112½.

Examination.—Physical examination at that time showed a large-boned, well-developed, muscular woman, with nothing abnormal in the head, neck, or chest, but with an extraordinarily relaxed anterior abdominal wall. The large scar which you see, which looks as if she had fallen sidewise on a circular saw, is what remains from the operation on her right kidney. I assume that the kidney was stitched in place, but it was very easy to feel it with its lower pole about three or four fingerbreadths below the level of the umbilicus, a little enlarged and very tender.

x-ray examination showed both stomach and colon to be very markedly prolapsed, and there was considerable general tenderness over the entire abdomen.

The patient stated voluntarily that when she went to bed her symptoms would almost invariably disappear during the period of her stay there. The blood showed a mild chloranemia; the urine was negative.

Diagnosis.—In regard to the diagnosis in her case, the first point to be noted is that we had found absolutely nothing of an organic nature aside from the manifest splanchnoptosis. The real question to be decided was whether the symptoms of which she complained, which, as you remember, were tolerably vague, with the exception of a pronounced loss in weight, could be fairly attributed to the splanchnoptosis. As I said to you before, this is a question to be decided in each case for itself. The patient's husband stated that she was a woman of great equanimity of disposition, was not in the least neurotic, and from what I could learn of her in the week I devoted to her examination I came to the same conclusion. An important point, it seemed to me, was the fact that all of the symptoms dated from shortly after the birth of her second child. I concluded, therefore, that the probabilities were that there was a definite relationship between her splanchnoptosis and her symptoms, and advised a long rest in bed, with overfeeding and exercises in the same way as in the

case of the patient I just showed you. In this patient's case, by referring to the history, I see that I did not treat her with the hourly feedings of milk and cream as I so often do, but instead gave her three regular meals a day, and then gave her milk and cream five times daily—immediately on waking in the morning, one hour after finishing each meal, and just before bedtime. I began with a 6-ounce mixture of milk and cream, increasing it later to 8 ounces. I can rarely ever remember having a more satisfactory patient. Her appetite picked up very rapidly, and in spite of the heavy diet she was getting she declared that she was constantly hungry. The course of her treatment was exceedingly uneventful. She gained weight steadily, and I dismissed her, after being in the hospital for seventy-six days, weighing 149 pounds, a total gain of 36½ pounds. She has not weighed herself recently, but in the time which has elapsed since passing from my care she has gained about 10 pounds more. I have seen her at intervals of perhaps five or six months, and she has remained in the very best of health, and she says now that her only trouble is that I trained her to eat so much that she is afraid of getting too stout. But she really weighs now very little more than at the time she was married.

Now let me make a few remarks on the question of operation in cases of this sort. I can express my opinion in a very few words. It is utterly irrational and therefore inadvisable. There is perhaps one group of cases in which operation may be warranted and may perhaps accomplish something, and that is in those cases which are congenital, and where the kidney is actually greatly displaced, *the other organs being in approximately normal condition;* in other words, where there is a real floating kidney, as an isolated phenomenon, and not dependent upon relaxation of the anterior abdominal wall. If scientific medicine means anything at all it means the treatment of causes and not of symptoms, and merely taking a kidney which is prolapsed as part of a general splanchnoptosis and anchoring it to a rib, even if the operation were successful in its results, is not striking at the root of the trouble. In this patient it accomplished nothing except to subject her to a needless operation.

The third patient I have to show you is again a case of high-grade splanchnoptosis with, however, one peculiarity—the patient is unmarried; and yet I have every reason to think that the condition is not congenital. Her history in brief is as follows:

Past History.—She had the usual diseases of childhood, and, in addition, a bilateral purulent otitis media at the age of seven and an acute rheumatism at the age of twelve. At thirty-three she had a severe attack of malaria, from which she recovered entirely. She was operated twelve years ago and both ovaries removed, but she does not know what the pathologic condition was. After this she was well for ten years.

Present Complaint.—For the past year she had been losing weight rather rapidly, having lost in the neighborhood of 20 or 25 pounds in that time. She had lost some weight even before that. A large part of the weight had been lost in the previous two or three months, during which time she had been under the care of a homeopathic physician, who put her on a greatly restricted diet. She went to him complaining of indigestion, "gas on the stomach," and obstinate constipation. She had noticed that she felt comparatively well in the morning on getting up, but after being up for a few hours commenced to feel worse, and at night her back would ache a great deal, she would complain of distress in the right side, and would have to lie down. She had never had headaches or vomiting. The gastric secretory functions showed nothing abnormal except a higher percentage of free hydrochloric than usual. The motor function was approximately normal, which is rather worthy of note, in view of the very low position the stomach was in at that time. The stools were absolutely negative, as was also the urine. She weighed 100 pounds when she came under my care, her normal weight being approximately 140 to 145.

Diagnosis.—Inasmuch as examination showed her to be of normal body frame, and inasmuch as there had been no traumatism of the anterior abdominal wall by pregnancy, it seemed to me that the most important factor in her splanchnoptosis was the loss of the intra-abdominal fat, which was largely due to her own injudicious attempt to "diet herself," and also to what seems to

me to have been ill-advised medical treatment. The small scar from the previous operation was perfectly healed and this was undoubtedly a negligible factor. I explained the situation to her, and recommended substantially the same treatment as in the two patients I have already shown you.

Treatment.—We carried out the treatment along the same lines, beginning in this case with a 3-ounce mixture of cream and milk hourly during her waking hours, gradually adding other liquid and semisolid food until she was gaining steadily. Owing to circumstances over which she had no control she could only remain in the hospital about ten days, but on removal to her home was able to carry out the same treatment there. She was in bed from October 7th until Christmas, 1912, and during that time gained approximately 40 pounds. She has been an exceedingly good patient since then and has kept up her exercises all the time. She insists that she feels very much better when she does them at least once daily, and she thinks they have a marked influence on her constipation. As long as she does the exercises she has no trouble with her bowels. She feels perfectly well in every way and has had absolutely no return of her trouble. She tells me that what she regards as the greatest gain is that, whereas before coming to me she was afraid to eat and her physicians kept her on a low diet, she finds that she can eat three good meals a day and feel perfectly well.

Now, gentlemen, in showing you these patients I have deliberately picked out patients who came under my care several years ago, and whom I have been able to keep under observation up to and including the present time. My reason for doing this is that so frequently patients are shown as good results a week or two or perhaps a few months after treatment, whereas continued observation shows that the results are not so good as were at first supposed.

Now let me caution you in regard to *after-treatment*. I warn these patients that *the important thing for them to do is to keep their weight at about what is normal for them*. So many factors result in loss of weight that I regard it as very important to have these patients weigh themselves from time to time, and see that

Fig. 114.—Stomach from Case 3. Horizontal position before undergoing treatment.

their weight is kept at the point which I consider normal for their individual case. I try to have them continue their exercises for

Fig. 115.—Stomach from Case 3. Horizontal position after undergoing treatment.

at least a year. It goes without saying that greatly weakened muscles cannot be restored to their normal tone in a few weeks.

Fig. 116.—Stomach from Case 3. Vertical position before undergoing treatment.

Fig. 117.—Stomach from Case 3. Vertical position after undergoing treatment.

This is sometimes productive of very interesting results. I have another patient who when leaving the hospital was in splendid

condition. I asked her to report in a couple of months, but she did not do so. In about four months her mother brought her in, saying that she was greatly discouraged because she had lost a great deal of weight. I asked her if she was feeling well; she said she was, and that she had been playing a great deal of golf and taking her exercises with great vigor twice daily, but that she had lost weight until her waist measure was 4 inches smaller than it had been. On looking at her waist-band it did seem as if it were at least 3 inches smaller. I then weighed her, for she had not weighed herself, and found that she had *actually gained 4 pounds*. She had jumped at the conclusion that she had lost weight because of the flattening out of the abdomen. I explained to her that the taking of so much exercise had resulted in the marked improvement in the tone of the muscles of the abdomen, and that this had in turn resulted in the lessening of girth. I have frequently seen patients who, while their weight remained practically stationary, would, in the course of a year, lose a couple of inches in girth from the exercises, but I have never seen as marked a case as the one to which I have just referred.

Now, in conclusion, let us sum up the situation as follows: Acquired splanchnoptosis is productive in some cases of symptoms; in other cases it is not. The first point to determine is whether the symptoms can be reasonably attributed to the splanchnoptosis. If so, the condition becomes a proper one for treatment. The indications for treatment are: First, to remove the traction on the organs by the recumbent position; second, systematic overfeeding to the point where the patients regain their normal weight; third, exercises designed to strengthen the anterior abdominal wall. It is, of course, self-evident, where we have a real floating kidney the result of an imperfectly developed thorax and abdomen, that not much will be accomplished by this method of treatment, but in my experience such cases are much less frequent than the acquired cases such as I have shown you. It would be hard to find three patients who are more grateful than these three, and as the treatment is simplicity itself, and can be carried on by almost anyone anywhere, I recommend it to your consideration.

CLINIC OF DR. FREDERICK TICE

COOK COUNTY HOSPITAL

A PERICARDIOMEDIASTINITIS SECONDARY TO AN ACUTE PLASTIC PERICARDITIS

Exploratory Pericardial Puncture. Types of Pericarditis. Association with a Double Mitral Lesion and a Right-sided Pleural Effusion.

April 19, 1916.

THIS patient was in the clinic once before, and at that time demonstrated as an acute plastic pericarditis. She had a very characteristic pericardial rub heard over the entire cardiac area; she presented cardiac manifestations secondary to a tonsillitis. She left the hospital materially improved. She remained at home only a short time, and returned to the hospital on the 5th of this month.

The *history* is that she returns with the same complaints as when previously admitted, consisting of precordial pain with difficulty in breathing, shortness of breath with a slight cough, and loss of appetite. Her previous history consists of a tonsillitis with a complicating pericarditis, as already indicated. Her temperature the afternoon she returned to the hospital was 102° F.; the following morning it was 101° F., and that afternoon, 101.8° F. The next day, on the 7th, the temperature in the morning was 101° F., in the afternoon, 101.4° F. On the 8th the morning temperature was 103° F.; in the afternoon, 100° F. On the 10th the morning and afternoon temperature was 100° F. Since that time it has fluctuated between normal and 101° F. This morning it is 100° F.

Physical examination reveals a definite increase in the cardiac impulse. The entire left chest moves with each contraction. There is a general heaving; but, on closer inspection, a localized

impulse is observed in the fifth intercostal space just outside of the nipple line. This impulse, the apex-beat, presents a systolic bulging; that is, with each contraction of the heart (when timed by palpation over the heart) there is a bulging with the systole, followed by a retraction. On palpation there is a strong, forcible cardiac impulse, no thrill over the apex or base. On percussion the right border of the heart extends well beyond the normal limit, midway between the sternum and the nipple line, running downward and outward to the hepatic dulness. On the left side the dulness is approximately at the anterior axillary line.

On auscultation over the cardiac area no pericardial rub can be heard at this time. Over the apex, which is near the normal location, a loud presystolic-systolic murmur can be heard; over the base of the heart there are no murmurs. The cardiac tones are tolerably distinct.

With the patient in an upright position there is impaired resonance over the lower half of the left thorax posteriorly. Bronchial breathing is present to the inner side of the lower angle of the left scapula. On the right side there is dulness on a level with the lower angle of the scapula, with a corresponding dulness anteriorly. This area of dulness shifts with change of position of the patient. Tactile fremitus and breath sounds are present.

Diagnosis.—The presystolic-systolic murmur is suggestive of a double mitral lesion, which may be either organic or secondary to a pericardiomyocarditis. The most important question is concerned with the possibility of a pericardial effusion. From the triangular outline of the cardiac dulness with the base directed downward, with the history of a recent acute plastic pericarditis secondary to acute tonsillitis, it would be natural to conclude that the patient is suffering from a pericarditis with effusion.

In addition to these findings there are signs in the left chest, posteriorly, indicative of compression. The *x*-ray plates show a definite blurring in the lower part of either the right or left side of the chest, more marked on the right, where signs of fluid can be demonstrated. The cardiac shadow is slightly increased, to the left of which is a fainter shadow, extending outward into the left chest.

An exploratory pericardial puncture was made, but no fluid was obtained. The puncture was made in the fifth intercostal space, a little to the inner side of the apex-beat. The point of the needle was constantly engaged. It was forced through the intercostal space to a sufficient depth to justify the belief that it must be in the substance of the left ventricle, but no fluid whatever could be obtained.

A puncture of the left chest, posteriorly, below the angle of the scapula, was negative. Aspiration of the right chest was done and 90 c.c. of serofibrinous fluid removed. The examination revealed a specific gravity of 1015, with many leukocytes present. The cultures showed a growth suggestive of a pneumococcus.

If this is not a pericardial effusion, associated with a double mitral lesion as a result of an acute infection, then what is the condition, and how are we to account for this increase in the cardiac dulness as indicated on percussion and in the skiagraph? A pericardial obliteration and adhesive pericarditis or a pericardiomediastinitis is frequent after an acute plastic pericarditis. The condition is divided into two types—the internal and external pericarditis.

In the former the pericardial sac is simply obliterated; no symptoms or signs are usually present. It is in the latter or external type, with an obliterated sac and an accompanying mediastinitis, that symptoms and signs are present. Where the adhesions are located in the pericardium only a great thickening may result. In a patient with pericarditis examined recently there was thickening of the pericardium amounting to 1 or $1\frac{1}{4}$ inches. During life she was constantly watched for signs of an obliterated pericardium, but none occurred. The autopsy, however, revealed a great thickening within the pericardium. Just such a condition may be present here, combined with a double mitral lesion and a right-sided pleural effusion, probably inflammatory, as a culture has been obtained from the aspirated fluid.

PULMONARY ABSCESS FOLLOWING DELAYED RESOLUTION IN A CROUPOUS PNEUMONIA

Etiology and Diagnosis. Method of Draining.

April 19, 1916.

Present History.—This patient entered the hospital twelve days ago. She is nineteen years old, a housewife, and was admitted to the surgical service. Her complaint at that time consisted of a pain in the lower portion of the abdomen, associated with a very severe and persistent cough, constipation, and a vaginal discharge, profuse night-sweats, and attacks of vomiting.

The pain in the abdomen has been present for one month, since an attack of supposed pneumonia. For the last two weeks she has suffered from a pronounced and persistent cough. This is associated with a dull, aching pain in her side which is aggravated and intensified by coughing. The cough has been present since the attack of pneumonia and is gradually becoming worse. She has had four pulmonary hemorrhages in the past two weeks; the first at night while coughing very severely, and at that time she expectorated half a cup of bright red blood. The other attacks came on while she was up and about. No vomiting or pain in the epigastrium was associated with these hemorrhages. Night-sweats have appeared and become worse in the past month; also a general weakness, particularly marked in the last two weeks. This is more pronounced in the afternoon, at the time of the onset of what she believes is a fever. The vomiting when it occurs is induced by the paroxysms of coughing. The vaginal discharge has been present for approximately three years; the onset was sudden and abrupt, but she denies that it followed an infection.

Personal History.—Menstrual history is negative. Since the age of thirteen or fourteen she has suffered from attacks of generalized convulsive seizures. This condition has been diagnosed as

epilepsy. She cannot tell when she is about to suffer one of these attacks; she is completely unconscious. The attacks lasts from a few minutes to twenty or thirty minutes, and afterward she does not know that she has had a convulsion until so informed. She also gives a history of having had pneumonia one month ago, which was preceded and accompanied by sore throat. There is a history of scarlet fever and measles in infancy. Her mother is living and well. Her father is living, and, so far as she knows, suffers from no disease.

When she entered the hospital on April 7th she was admitted with the diagnosis of "pyosalpinx." The physical examination at that time revealed, so far as is recorded here, definite findings in the pelvis, substantiating not only the diagnosis of a double pyosalpinx but also a retroversion and flexion. In addition to this, there were signs indicating a thoracic involvement.

When the patient was admitted she had a temperature of 102° F., a pulse of 128, respirations 26. The following morning the temperature was 99° F., but that afternoon it reached 101.8° F. On the following day the morning temperature was 100° F., in the afternoon it was 102° F., and at that time she was complaining of pains, particularly in the upper portion of her abdomen. On the tenth her morning temperature was 101.6° F., and that afternoon it reached 103° F.; on the eleventh in the morning it was 101° F., in the afternoon, 103° F. On the twelfth in the morning it was 99° F., and that afternoon, 102° F.; on the thirteenth in the morning, 100° F.; in the afternoon, 102° F. At this time the patient was transferred to the medical ward on the diagnosis of an unresolved pneumonia associated with the pelvic condition as already indicated.

Since that time she has presented almost continuously a morning temperature that is approximately normal, but in the afternoon it reaches 102° to 103° F. or a fraction above. The white count when she was admitted was 15,000; on the fourteenth a second white count was made which was 19,600. The urinalysis made the first morning she was admitted was negative. A second one, on April 15th, was also negative.

Physical Examination.—You have all taken note of this rather

pronounced paroxysm of coughing from which the patient is now suffering. At times, for two or three hours, she is perfectly free; then a paroxysm will occur, lasting for a few minutes, followed by considerable expectoration, sometimes coming up with a good deal of effort and then again without much exertion. Associated with these attacks of coughing there is a rather definite, pronounced odor, but not particularly offensive. There is a history, as given when admitted, of pulmonary hemorrhages, and on this account a tentative diagnosis was made of a pulmonary tuberculosis, and this is one of the conditions which must be considered in the diagnosis.

Due to this attack of coughing the patient is perspiring freely, as is evident on the forehead, face, chest, and abdomen. She also has, as you can observe, a fair degree of anemia, judging from the cutaneous surface and the mucous membranes of the mouth and the eyes. At present the patient is able to assume the dorsal position, but during the attack of coughing she was on her left side, and when requested to turn on her right side she replied that she could not. That must be taken into consideration because it may have some bearing on the physical findings and the diagnosis. You will also recall that when she sat up the coughing became less. These are all points which must be considered in arriving at a diagnosis. On inspection, aside from the character of the breathing, which is rapid and rather superficial, there is apparently but very little difference in the size or contour of the two sides of the chest. If anything, possibly the right side is a little larger and the intraclavicular region somewhat more prominent, but the expansion is approximately equal. On the left side there is a very pronounced diaphragm shadow, or Litten's sign, but it is absent on the right. To this we will refer again in connection with the fluoroscopic examination.

On percussion of the chest anteriorly there is a good resonance on the left side from well above the clavicle downward to the normal splenic border, but the resonance is not so good on the right; in other words, there is a relative dulness or impaired resonance on the right or a hyperresonance on the left.

The cardiac dulness: The right border is approximately at the midsternal line, the upper border at the third rib, and the left border one finger outside of the midclavicular line.

Percussion posteriorly on the left side gives a good resonance, extending from the apex down to a full handbreadth below the scapula. On the right side there is a good resonance over the apex of the lung, but on reaching a point on a level with the spine of the scapula dulness is encountered which increases and becomes practically flat. The outline of this area of dulness corresponds quite accurately to the outline of the right lower lobe of the lung. Over this area, as before indicated, the percussion resonance increases from above downward, so that it becomes practically flat in the lower portion.

Palpation over the anterior and posterior surfaces of the left thorax is normal. Over the right chest and above our line of dulness the tactile fremitus is slightly increased, but over the lower portion, over the area of dulness, the tactile fremitus is almost absent.

On auscultation over the entire left lung the respiratory findings are normal. Over the right chest above our line of dulness the breath sounds are slightly intensified, but not associated with râles. Over the area of dulness the breath sounds are definitely tubular with practically no râles. Occasionally an inspiratory râle is present. The voice sounds are well transmitted over this area of dulness. Aside from the rapidity the heart is negative.

Diagnosis.—Having heard the clinical history and determined the physical signs, the next problem is concerned with the diagnosis.

DOCTOR: I would suspect a pneumonic phthisis.

DR. TICE: What else?

DOCTOR: Delayed resolution.

DR. TICE: Yes; what else?

DOCTOR: A chronic tuberculosis secondary to pneumonia.

DR. TICE: Yes; has anyone else a suggestion?

VISITOR: A bronchial pneumonia with a pleural effusion.

DR. TICE: Yes; simple or purulent?

VISITOR: Simple, encapsulated.

DR. TICE: Anything else? There are other conditions here.

DOCTOR: A gangrene.

DR. TICE: Yes; you have all smelled this definite odor. That suggests a possibility which must be taken into consideration. An abscess is a strong possibility here for the very reason that we have the history of a pneumonia, since which time the patient has not been well, and she entered the hospital with a temperature which is nearly normal in the morning and goes up in the afternoon to 102° or 103° F., a leukocytosis, and indications of a purulent condition somewhere.

Our physical findings indicate an involvement on the right side of the chest. Some of these conditions mentioned we can touch upon and practically disregard them, but there are a few which we must give consideration, remembering the possibility of some complication following pneumonia.

If the patient's statement is correct, that she had pneumonia before she entered the hospital, it becomes necessary to consider some of its complications. Of these there has already been mentioned the possibility of a delayed resolution, a condition previously demonstrated here in the clinic, and which is by no means rare. Other possible complications consist of an empyema, either a free empyema (the pus in the pleural cavity) or an encysted empyema (the pus walled off by adhesions), or an interlobar empyema (where the pus has become encapsulated in one of the fissures), or a pulmonary abscess associated with gangrene. You will recall the pathologic specimen presented here in the clinic of a case of gangrene following pneumonia in which the lower lobe was gangrenous, and the patient died with an acute pulmonary hemorrhage.

It is also necessary to determine whether or not this is tuberculosis, either primary or a tuberculosis following pneumonia. We have some pretty good evidence that tuberculosis is one of the conditions to be considered.

First of all, so far as can be determined, the onset of this pulmonary involvement was abrupt and sudden, an onset rather suggestive of a croupous pneumonia, but it must be remembered that an acute pneumonic phthisis may come on with a chill. The

location of the involvement is against a tuberculosis, while a lower lobe may be primarily involved in acute pneumonic phthisis; as compared to an upper lobe it is relatively rare. There is also a leukocytosis, which is more suggestive of some other infection than a tuberculosis. Repeated examination of the sputum has been negative. The family history is negative as to tuberculosis.

There are a few other conditions which we must seriously weigh. First of all, an empyema must be considered, and here an encysted empyema, for the reason that there is no movable area of dulness; when we change the position of the patient the area of dulness remains the same. The tactile fremitus is retained and the breath sounds are definitely tubular. All of these findings speak rather strongly against the existence of an encysted empyema. A puncture was made as a means of diagnosis, to which we will refer later.

Another possibility is an interlobar empyema, but the outline of this area of dulness corresponds quite accurately with that of the right lower lobe. The area of dulness is below the interlobar fissure.

Now, as to the possibility of gangrene: It is true this patient is expectorating offensive material, and she has had definite hemorrhages, which are not infrequent in gangrene, but the character of the sputum is not suggestive. In a gangrene the sputum is extremely offensive, the amount expectorated considerable, and when allowed to stand it will separate into three definite and distinct layers. The examination of the sputum also fails to reveal the presence of elastic or pulmonary tissue.

When this patient was first examined the conclusion was that she had a delayed resolution.

The patient was sent to the *x*-ray department for examination, which showed that the right lower lobe was infiltrated. No fluid could be demonstrated in the pleural cavity. There was no indication of a pulmonary cavity or anything other than an infiltration or delayed resolution.

On account of the chills, fever, and leukocytosis a puncture was made over the area of dulness, the needle at first being directed almost in a straight line, which gave a dry tap. The

needle was then withdrawn and inserted upward, and possibly 2 drams of a thick, purulent fluid were obtained. Examination of this fluid revealed the presence of a pure culture of a pneumococcus. This brings up the consideration of a pulmonary abscess. Is this an encysted empyema or a pulmonary abscess, or an unresolved pneumonia with abscess formation, or a possible beginning gangrene with abscess formation?

In the patient presented a couple of months ago the first puncture gave a dry tap. A few days later another puncture produced about a dram of pus, and on the third occasion repeated punctures gave only dry taps, but the gross specimen revealed a gangrene with multiple abscesses through the entire substance of the lung.

This patient, from the standpoint of an abscess of the lung, presents the usual history and symptoms; that is, an attack of pneumonia followed by an irregular temperature, a leukocytosis which has increased since she entered the hospital, profuse attacks of sweating, and paroxysmal attacks of coughing with a foul, offensive sputum and breath. Perhaps the most frequent symptom is a paroxysm of coughing and the sudden expectoration of a large amount of pus, following which the physical examination may reveal the findings of a pulmonary cavity.

The lack of a diaphragm shadow, the failure of the fluoroscopic examination to demonstrate diaphragm movements or to demonstrate the presence of movable fluid in the right chest, and the depth to which the needle was inserted in obtaining the pus, would indicate a pulmonary abscess.

Etiology.—A pulmonary abscess is associated with a great variety of conditions. Perhaps one of the most frequent is lobar or croupous pneumonia, and this is quite liable to occur where the resistance of the patient is low. An abscess is not infrequent in bronchopneumonia, and perhaps more frequent when associated with a malignancy, as a carcinoma of the mouth, where the purulent material is aspirated into the lung. It is a not uncommon sequel to pulmonary emboli. Empyema not infrequently ruptures through the pleura and results in abscesses. Tuberculosis is a frequent cause of pulmonary abscess. A ruptured bron-

chial gland or a suppurative mediastinitis or suppuration extending through the diaphragm, as from appendicitis, or a liver abscess may result in pulmonary abscess. In this instance a croupous pneumonia with delayed or imperfect resolution resulted in an abscess. In this condition the abscess may be single or multiple.

Prognosis.—In a series of 25 cases of pulmonary abscess here in the County Hospital 23 made a perfect recovery.

Treatment.—After making a puncture and demonstrating the presence of pus, as was done in this instance, and particularly if the patient shows marked symptoms of infection, the best plan is to consider the condition surgical and to recommend operative interference. This puncture was made on Saturday, two days ago. Her condition today is not so favorable as at that time, and as a result she will be transferred to the surgical service, with the request that this abscess be drained.

The method of operation is always an important consideration, particularly as to the question whether the pleural cavity is obliterated or not. It is, comparatively, a simple matter to open a pulmonary abscess if the pleural cavity is obliterated by adhesions, for there is little or no danger of collapse of the lung, but it is often a difficult problem to determine whether the pleural cavity is free or obliterated. In this patient the physical signs indicate that the pleural cavity is obliterated, but the safest procedure is to fix the pleura before the incision is made into the pleural cavity. In other words, fasten the lung by three or four sutures and then make the incision at once or after a lapse of twenty-four to thirty-six hours. This opening is usually best done by making a preliminary puncture and locating pus, leaving the needle in position, and making the opening, preferably with 8-inch forceps, along the side of the needle.

The Paquelin cautery has been advocated and is used for this purpose, but the results obtained make its use inadvisable. A good deal of necrosis always ensues and an incision is much to be preferred. A tube must be left in place, and most of the pus will be evacuated promptly and the patient go on to perfect recovery.

Subsequent Note.—The young lady, with the diagnosis of "pulmonary abscess," was referred to the surgical side and a rib was resected. The pleural cavity was found completely obliterated. No pus escaped after the resection, and at first there was some doubt about the presence of an abscess. A puncture was made, pus obtained, and an opening was made into the pus cavity which was situated 1½ inches below the lung surface. Immediately after the operation the patient was seized with an attack of coughing and expectorated a large amount of blood-tinged, offensive material.

RECTAL STRICTURE FOLLOWING OPERATION FOR HEMORRHOIDS

Differentiation from Carcinoma of the Rectum and Syphilitic Stricture.

Present History.—This patient was sent into the hospital March 18th with a diagnosis of "dysentery." She is thirty-six years of age. One year ago she began to have a pain in the lower part of the abdomen with tenesmus. Six months ago she was compelled to go to a hospital on account of diarrhea and loss in weight. She has been in bed the last six months. Her bowels move on an average of five times a day, and often as much as twelve times a day. There has been great loss in weight, particularly during the last six months. There has been no cough and no vomiting. She has at times severe pain in the small of the back. The pains in the abdomen come on at intervals.

Past History.—She was never sick before the onset of the present trouble. Her father and mother are living and well; one brother and two sisters are well. Her temperature varies from normal to 99° or 100° F. The pulse varies from 80 to 110. The respirations are normal. The urinalysis is negative. A blood examination reveals a white count of 8900, a red count of 3,340,000, and a hemoglobin of 40 per cent. Examination of the blood-smears shows 71 per cent. polymorphonuclears and a secondary anemia.

The *examination* of this patient, aside from the anemia, which is quite evident from the external surface of the body, as well as the mucous membranes, reveals but very little, with the exception of the rectal examination. There is no distention of the abdomen and no tenderness, but the patient complains of abdominal distress and pain at intervals. A little tenderness is elicited in the hepatic region. What possibility does that suggest?

DOCTOR: It might be a hepatic abscess if she has dysentery.

DR. TICE: What examination is essential to establish the existence of an amebic dysentery?

DOCTOR: Examination of the stools for Amœba coli.

DR. TICE: Repeated examination of the stools have revealed no ameba. A single examination may not be successful, but repeated examinations will almost invariably reveal the presence of the ameba if a dysentery exists. The Weber is positive. She is passing a very considerable amount of blood.

The stool examination, while important, does not complete the examination or fulfil the desired requirements. The usual or instrumental rectal examination is necessary.

Digital examination reveals a rectal stricture just inside the sphincter. It is smooth, no ulceration or granulations are present, and it will only admit a small finger.

DR. TICE: What are some of the possibilities?

DOCTOR: A carcinoma or a stricture due to an infection or scar-tissue from other causes.

In cases of suspected carcinoma of the rectum it is well to keep in mind the possibility of a syphilitic stricture. There is no syphilitic history in this patient, but, as a matter of routine, a Wassermann was done and reported negative.

In doubtful cases of carcinoma a small piece of tissue should be removed for microscopic examination.

This patient's history is of considerable significance in arriving at a diagnosis. She first had hemorrhoids, which were operated, and her present condition followed. This is a stricture following surgical interference for the hemorrhoids. The treatment is surgical, and this patient will be transferred to the surgical side for operation. Probably only mechanical dilatation will be necessary.

AN ABDOMINAL ANEURYSM

Discussion of the Physical Signs with Special Reference to Differential Diagnosis. Pathogenesis of Aneurysm. Frequency of Positive Wassermann Reactions in Cardiovascular Disease.

April 15, 1916.

Present History.—This patient is forty-three years of age and entered the hospital on March 24th, with the history of rheumatism four years ago, which entirely disappeared and has not recurred. About one month ago she was seized with an attack of choking and smothering, which came on at night and interfered particularly with her breathing. These attacks occurred at irregular intervals and lasted for a few minutes. The dyspnea began with exertion, and now she can hardly walk. She is unable to lie flat, and this has been more or less marked during the past three weeks.

Previous History.—Aside from the attack of rheumatism, as already noted, she had the ordinary diseases of childhood. The family history is negative. She gave birth to one child which died at the age of five. No definite cause is known. There is no history of miscarriages or abortions; the venereal history is negative according to her statement. There is no history of the use of alcohol.

Physical Examination.—The statement was made that the patient has a cardiac lesion associated with a tumor in the abdomen, chiefly in the epigastrium. Upon inspection in the region of the heart we observe an increase in the cardiac impulse, very definite in the third, fourth, and fifth interspace. The impulse, particularly at the apex, is displaced outward—practically in the nipple line. We also observe a very definite pulsation in the suprasternal notch, in the supraclavicular and cervical regions, and a pronounced pulsation in the axillary, in the brachial, and even down into the radials.

On palpation, there is a very strong forcible cardiac impulse, the apex in the fifth interspace is increased in area, requiring at least three finger-tips to cover the impulse. There are no thrills over the apex or over the base of the heart. On percussion, the right border of the heart extends over two fingers in the fifth intercostal space, and one full finger in the second intercostal space, to the right of the sternum. The left border of the heart extends out to the axillary line. The base of the heart is at the upper border of the second costal cartilage; the aortic dulness extends one finger to the left of the sternum in the first intercostal space.

On auscultation over the apex of the heart there is a distinct presystolic-systolic murmur. The first sound is not accentuated. Above, in the fourth intercostal space, there is a double murmur, a systolic and diastolic, which gradually increases in intensity as the base of the heart is approached, and reaches its greatest intensity in the region of the aorta. These murmurs can be heard in the upper portion of the chest and over the large vessels of the neck.

From these considerations, aside from the peripheral findings, it is a safe conclusion that the patient has an endocarditis. The diagnosis of an aortic insufficiency can be confirmed on the basis of the increased cardiac dulness, the diastolic murmur heard with maximum intensity over the base of the heart, associated with visible pulsations of the vessels of the neck and upper extremities. Further examination reveals the presence of a typical Corrigan and Quincke pulse with a Duroziez's sign. The systolic murmur over the base of the heart is not so easily interpreted. The three cardinal findings of an aortic stenosis, consisting of a systolic murmur over the base of the heart, a systolic thrill in the same region, associated with a decrease in the volume of the radial pulse, are not present. There is only the one, the systolic murmur, and this finding is so frequent in other conditions, as in atheroma, an aneurysm, or a dilatation of the aorta and in the mere roughening of the valves without a definite stenosis, that it is not possible to make a diagnosis of an aortic stenosis. There is an increase of the dulness over the aortic area and other indications to justify the belief that the aorta is dilated.

The presystolic murmur at the apex is not associated with a thrill or with an increased first sound. A presystolic murmur of this character, associated with an aortic diastolic murmur, is probably functional in origin or an Austin Flint murmur. It is due to the regurgitating stream of blood from the aorta into the ventricle impinging on the mitral leaflet, which interferes with the flow from the auricle into the ventricle. Or perhaps the murmur results from an overdistention of the left ventricle from the regurgitated blood from the aorta, which dilates the mitral orifice, puts the mitral leaflets on a tension, and interferes with the free flow from the auricle to the ventricle.

The systolic murmur at the apex indicates a mitral insufficiency. By inspection, the tumor mass which is present in the epigastrium, situated to the left of the median line, is easily the size of a large orange. There is a definite pulsation, which is synchronous or nearly synchronous with the cardiac beat. There is not only an up-and-down pulsation, but, when palpated, a pronounced expansive pulsation. No thrill is present over the tumor, but there is a thrill in the median line to the right of the projecting mass. The thrill is easily palpable as low down as the umbilicus. The tumor is fixed, cannot be moved.

On palpation it is easily determined that this tumor mass is much larger than appears on inspection. Light palpation determines the right border at least three fingers to the right of the median line. It extends at least a finger below the umbilicus. The left border extends under the costal border, making a tumor mass at least the size of a fetal head, with a bulging portion in the left hypochondrium. The deeper mass possesses a very definite pulsation; only a single impulse can be determined.

On auscultation, a relatively faint systolic murmur is audible. Over the region of the aorta in the median line and just to the right of the tumor a very distinct, relatively loud systolic murmur is heard. This is audible as far down as the umbilicus.

With these findings, it is necessary to consider some of the possibilities.

A tumor located in this region would necessitate at least the consideration of a carcinoma of the stomach or the pancreas, a

neoplasm of the liver or a gumma of the liver, an omental tumor, or a neoplasm originating from some other locality. The mere fact of its being expansible does not establish the existence of an aneurysm. A rapidly growing sarcoma or even benign tumors situated in the region of the aorta may produce an expansile pulsation.

Several of these considerations can at once be reasonably excluded, particularly when we consider some of the evidence in favor of an aneurysm. A definite expansile pulsation, associated with a thrill and a murmur over the region of the aorta, and with the further findings in the heart, will at least incline our opinion to the presence of an aneurysm.

Some further evidence in favor of an aneurysm was obtained by a fluoroscopic and skiagraphic examination. The fluoroscopic report is that there is a definite expansile pulsation, particularly when the patient is placed in a lateral position. The independent pulsation of the heart and the tumor could not be determined. A skiagraphic examination was made to ascertain the relation of the tumor to the stomach. The question was raised, at the time of one of the examinations, where the stomach was situated in relation to the tumor? To determine this a barium meal was given, which is represented in the skiagram. The dark area is the tumor mass; the stomach, containing some of the barium, is so compressed as to practically surround two-thirds of the tumor mass.

Further evidence in favor of an aneurysm is found in the examination in reference to the etiology. The venereal history was negative, but a Wassermann was made on the 25th, and the laboratory reported a double plus reaction. The patient was pregnant once and gave birth to a child that died at five years of age; further than that she has never been pregnant. The urine is negative. There is a leukocyte count of 5300. Before the Wassermann was made a specific infection was strongly suspected, particularly in view of the cardiac and aortic involvement and the clinical diagnosis of an abdominal aneurysm. The Wassermann report confirms this suspicion and strongly favors the existence of a syphilitic aortitis.

An aneurysm in this location is not unusual, and when discussing syphilitic aortitis the two locations of election were mentioned: First, in the ascending portion of the thoracic aorta, and, second, in the upper portion of the abdominal aorta. The favorite site is just above the celiac axis. The clinical diagnosis is an aneurysm of the abdominal aorta which takes its origin in or near the celiac axis. The diagnosis of a syphilitic aortitis and endocarditis, with an aortic and mitral insufficiency, is justified on the basis of the physical signs and a syphilitic etiology. The diagnosis of a syphilitic aortitis was discussed at a previous clinic, and at that time reference was made to the mode of production of an aortic aneurysm.

Pathogenesis of Aneurysm.—Briefly considered, our knowledge of aneurysm can be divided into three periods: The first period begins with the recognition of an aneurysm by Ambroise Paré. Not only did he diagnose but also observed that aneurysms occurred particularly in those who had undergone mercurial rubs or treatment for the so-called French disease. It would seem that Vesalius, while acquainted with aneurysms from his anatomic experience, entirely ignored the syphilitic etiology, but his contemporaries called attention to their syphilitic origin.

The second period consists of their clinical study by Dietrich and others. The third period, including the present time, dates back to 1905, when Schaudinn announced the discovery of the specific origin of syphilis. Then came the announcement of the Wassermann reaction, and four years later the discovery by Ehrlich of salvarsan.

Reference has already been made to the work by Chiari, published in 1903, in which he differentiated a syphilitic aortitis from an atheroma, but this was before the discovery of the specific organism or the Wassermann reaction. Since then the demonstration of the specific organism and the Wassermann have confirmed this finding and also the manner in which the specific infection produces an aneurysm; it is typically a mesaortitis.

First of all, there is an increase in the media, with destruction of the elastic fibers, and with this secondary changes in the intima and adventitia, which result in dilatation and the forma-

tion of an aneurysm. In another group the process may involve all three of the coats in a comparatively rapid process of weakening, with a local or diffuse bulging. The influence of intravascular pressure is of importance; any strain should be considered a contributing factor as increasing the force upon a weakened vessel.

The frequency with which a positive Wassermann is obtained in cardiovascular disease is noteworthy. A review of the published reports reveals the fact that 25 to 50 per cent. in aortic insufficiency and 75 to 100 per cent. in aneurysm will give a positive Wassermann. Wolfson has recently reported his investigations regarding the Noguchi test, and obtained 95 per cent. positive reactions.

Treatment.—The primary object is the treatment of the etiologic factor, *i. e.*, the specific infection. This patient is on iodids and mercury; salvarsan or neosalvarsan is indicated, and might produce some benefit. The treatment of the aneurysm consists in instituting rest, the application of ice-bags, and of surgical interference. In one of the aneurysms here in the hospital wiring was done, with the result that the patient lived for four or five months, but died from the result of an embolism. In another one of the patients, with an abdominal aneurysm of this type, a ligation of the aorta was done with a fatal outcome in twelve to fourteen hours. The majority of these cases die from a rupture of the aneurysm, usually into one of the neighboring cavities.

A CASE OF TRUE AMEBIC DYSENTERY

A Mild Type of the Disease Developing in Chicago. Emetin and Ipecac in the Treatment.

April 15, 1916.

Present History.—This patient is forty years old, and entered the hospital on March 31st with a complaint of diarrhea, averaging four or five movements a day, and extending over a period of six months. There is edema of the feet and legs, with loss in weight and appetite. The first symptoms appeared about six months ago, when he noticed an increase in the number of stools and some of them definitely bloody. Five months ago he entered the hospital and remained for two months, and left apparently not much improved. The difficulty with the bowels continued, he lost his appetite, lost weight, and finally cramp-like pains occurred in the iliac region. He was never sick until this present difficulty began.

Family and *personal history* are negative. This man has lived in Chicago for the last four years; he is a laborer; previous to his coming to Chicago he lived in Russia.

His temperature the day of entrance was 98.2° F.; since that time the temperature is reported as 97.8° F., and on one occasion 99° F.; this morning his temperature is 97° F. The pulse-rate during the first ten days was 84 to 88, then it fell to 60, where it has remained until this morning, and now it is 80 per minute.

Examination of the stools from this patient reveals the presence of the specific organism of amebic dysentery. The organisms were found in abundance the day after he entered the hospital. The leukocyte count is 10,900. The urine is negative. The physical examination reveals practically nothing. There is no distention or tenderness of the abdomen, no enlargement or tenderness in the region of the liver, only a little tenderness over the region of the descending colon.

The *diagnosis* of an amebic dysentery is of interest not only in itself, but from the fact that this patient has lived in Chicago for four years, and developed a bowel disturbance six months ago, which we now know to be an amebic dysentery. Not a few cases of true amebic dysentery occur in this locality. Individuals who have lived continuously in Chicago and have never been out of the city have been known to suffer from dysentery.

The patient, aside from the dietetic regulation, is receiving emetin hydrochlorid, $\frac{1}{2}$ grain hypodermically twice a day. This is the treatment which is being pursued in these cases at the present time. In the Philippines the use of emetin twice a day in this dose is recommended, then once a day for four days. This is followed by ipecac by mouth. The emetin will have little or no influence over the organism in the intestinal canal. When the organism is in the tissues the emetin is far more efficient than the ipecac, but when the amebæ are in the intestinal canal ipecac is to be preferred.

So far this patient has received, up to the present time, only four hypodermic injections of emetin. The prognosis, so far as can be determined, is decidedly favorable, from the fact that it is apparently only an intestinal dysentery of comparatively mild type.

CLINIC OF DR. FRANK WRIGHT

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

ACIDOSIS

Newer Methods for Detecting the Presence of Acidosis and for Determining Its Degree. Clinical Applications.

BEFORE presenting a patient with the clinical picture of acidosis I will discuss with you the subject and some of the newer methods for determining the degree of acidosis.

Under normal conditions in the course of metabolism many acid bodies are produced in the cells, leave the cell in the lymph, and are carried by the blood to the organs through which they are excreted. While in transmission they are not in solution as free acids, but in combination with basic substances. At the place of excretion they are, in part, separated from these bases, which are returned to again fulfil their function, while some of the acids are excreted with their bases in the form of salts. Examples of acid elimination are the excretion of CO_2 through the alveolar epithelium and the excretion of the fixed acids as acid salts through the kidney.

While the transportation of these acids is going on the blood maintains a reaction which is, to all intents, neutral, verging slightly toward alkalescence. This neutrality is maintained even under conditions which are adverse, and a change to an acid reaction is not compatible with life.

To emphasize this most striking maintenance of neutrality it has been aptly stated that the difference between the alkalescence of normal body fluid and an acidity which would cause death amounts to the difference between the alkalinity of tap-water and the comparative acidity of distilled water.

This being the case, we are not concerned, then, with the reaction of the circulating blood itself, but rather with its ability to combine with acids, on the one hand, or its reaction after being deprived of its volatile acid (CO_2) on the other. We want to know the extent of the alkali reserve, the amount of basic material which is available in the blood.

We may readily see that this reserve may be decreased by—

(*a*) An overproduction of acid bodies.

(*b*) A slow excretion; hence, an accumulation of these bodies in the blood, rendering a portion of the bases unavailable.

(*c*) Any condition in which the bases are excreted too rapidly or in excessive quantity.

(*d*) Lastly, in those conditions in which bases are not furnished the body or, if furnished, are not absorbed.

Approaching the subject from the standpoint of the alkali reserve as well as from evidence of acid production, we find a number of newer methods for estimating both which have a very practical clinical bearing. These methods may be classified in three groups:

I. Those which have to do with the estimation of the CO_2 content of the alveolar air, two of which are sufficiently simple to be of use clinically:

(*a*) Fridericia method.

(*b*) Marriott method for CO_2 determination.

II. Those methods which determine the reserve alkalinity of the blood, either directly or indirectly:

(*a*) Marriott's modification of the Levy-Rowntree and Marriott method for determining the residual H ion concentration of the serum.

(*b*) The Van Slyke method of estimating the CO_2 combining power of the plasma.

III. Method for estimating the acids and bases of the urine:

(*a*) Van Slyke's method for estimating ketonic acids.

(*b*) Attention to the quantitative amounts of base excreted, by which I refer to the estimation of calcium and magnesium as well as ammonia (NH_3).

In more detail, the alveolar air methods depend upon the fact that the tension of the CO_2 in the alveoli varies with the solution pressure of the CO_2 in the blood.

In conditions of acidosis the alkali reserve of the blood is low and the amount of CO_2 which combines is lessened; hence, a smaller quantity of CO_2 is found in the expired air. (Incidentally one might remark that increase in respiratory rate is one of the first things involved.)

Normally the tension is about 46 mm. of Hg. = 6 per cent. CO_2. Under abnormal conditions amounts corresponding to 15 mm. (less than 2 per cent.) are sometimes found, and anything under 30 mm. gives evidence of acidosis.

One is attracted by the simplicity of the alveolar air methods, and by the fact that air can be obtained without causing pain and as frequently as desired. However, there are many drawbacks, the greatest being the securing of a representative sample of air. All one has to do is to make a first attempt to secure samples by breathing into the apparatus or bag oneself to appreciate the difficulties involved. Also with children, with people of low intelligence, and with the patients who are very ill many trials and infinite patience are required.

Errors tend to lower the readings and so simulate the low readings which are of pathologic significance, so one must be sure of the accuracy of his results before he attempts to interpret them.

The **Fridericia method** aims to secure alveolar air with a tension corresponding to that of arterial blood. At the end of a normal inspiration air is exhaled through a tube of 100-c.c. capacity, and the latter portion of the expired air is held there by turning a stopcock. The temperature is brought to that of the room by immersing the tube containing the air in water of the same temperature, after which the CO_2 is absorbed with 20 per cent. potassium hydroxid solution, water is admitted to compensate for the loss in volume, and after readjusting levels and temperature the loss in volume is read directly on the graduated portion of the tube as the percentage amount of CO_2 of the expired air. This method will probably be superseded by—

Marriott's Method.—Here the CO_2 content of the alveolar air corresponds to the content of the venous blood, and so is higher than found by the Fridericia method.

The patient, with nose pinched with a padded clamp, at the end of a normal expiration (avoiding taking a deep breath) breathes the air through a mouthpiece attached to a rubber bag containing 600 c.c. of pure air. After breathing back and forth four times in twenty seconds the bag is clamped off at the end of the fourth expiration. This sample of air is then bubbled through a $\frac{N}{100}$ solution of sodium carbonate or bicarbonate containing 20 per cent. by volume of $\frac{1}{100}$ per cent. phenolsulphonephthalein, and the resulting color obtained is compared with that in standard tubes of known reaction and corresponding to those obtained by using air containing CO_2 of known amounts as expressed in millimeters of mercury. The test depends upon the balanced reaction or ratio between the alkaline bicarbonate and the acid CO_2 in the solution. This ratio varies with the varying amounts of CO_2 present in the air used for saturation, but in any case once the solution is saturated no more is taken up, no matter how much of the air mixture is bubbled through. This makes it a most valuable clinical method and one which can (in fact, *must*) be done rapidly, for the solution, if allowed to stand, soon loses CO_2, and so changes color.

Normal individuals show CO_2 with a tension from 40 to 45 mm. Hg., corresponding to 5.5 to 6 per cent. To transpose the amount of CO_2, as expressed in millimeters of mercury to percentage figures, one divides the reading in millimeters by the height of the barometer less the vapor tension, also given in millimeters, *e. g.*:

Alveolar air tension	40
Barometer less vapor tension	710

then $40 \div 710 = .056$, which again is equal to 5.6 per cent. CO_2.

The Methods Applicable to the Blood Itself.—(*a*) Marriott's method for determining the reserve alkalinity, which he designates the R_PH, depends upon the fact that buffer solutions can be diluted without appreciable change in their reaction, as ex-

pressed by their H ion concentration. The blood is a very good example of a fluid showing buffer action, and its power to neutralize acids without having its reaction altered from the very narrow physiologic limits is due to this property. Clear, fresh serum, free from hemolysis, is dialyzed through collodion sacs into normal (0.8 per cent.) sodium chlorid solution containing phenolsulphonephthalein as indicator. The dialysis is continued sufficiently long (seven minutes) to allow the reaction of the salt solution to reach that of the serum, after which the CO_2 is driven off by bubbling pure air through the solution. The reaction is then found by comparing the color with those of solutions of known reactions or H ion concentration. When acidosis is present the alkalinity is reduced, that is, the reaction more nearly approaches neutrality.

The difficulties met with are:

(1) Obtaining and maintaining a neutral salt solution.

(2) Preparing the collodion sacs.

(3) Getting serum free from hemolysis.

(4) The nomenclature involved.

The first three can be readily overcome, but the last requires considerable time to explain satisfactorily. One can take the figures as given, 7 representing neutrality, and figures higher than that indicating alkalinity, while lower figures are used for acidity; then the blood reaction falls between 7 and 8, which emphasizes how close to neutrality it is, but always during life remaining on the alkaline side. My findings normally are 7.8 to 8. If not satisfied with this explanation, we may go further.

At 25° C. water dissociates into its ions H and OH to the extent of 1 part of H (or 1 gram) in 10,000,000 liters, and 1 part of OH (= 17 gm.) in the same quantity—*e. g.*, 10,000,000 liters. We express this as C, meaning concentration, of H and OH, or $C_H^{-7} \times C_{OH}^{-7} = 1 \times 10^{-14}$. The 1×10^{-14} being another method of expressing one in 10,000,000.

If in any solution the amount of ionized hydrogen is increased the amount of ionized OH (hydroxyl) is correspondingly decreased, and the product obtained by multiplying the concentration of the H by the concentration of the OH remains constant; if one goes up the other goes down.

A solution which contains more ionized hydrogen than water does is an acid solution, and one in which the OH (hydroxyl) ion predominates is alkaline.

Then, going back to the formula given, $C_H^{-7} \times C_{OH}^{-7} = 1 \times 10^{-14}$, we can at least see where they get the figure 7 to indicate the neutrality of water where the H and OH are balanced. If I may follow this another step:

$_pH$ of	0 =	$\frac{N}{1}$	solution of acid.
	1 =	$\frac{N}{10}$	solution of acid.
	2 =	$\frac{N}{100}$	solution of acid.
	3 =	$\frac{N}{1000}$	solution of acid.
	4 =	$\frac{N}{10,000}$	solution of acid.
	5 =	$\frac{N}{100,000}$	solution of acid.
	6 =	$\frac{N}{1,000,000}$	solution of acid.
	7 =	$\frac{N}{10,000,000}$	= Neutrality. Having the same ion content as water.
	8 =	$\frac{N}{1,000,000}$	solution of alkali.
	9 =	$\frac{N}{100,000}$	solution of alkali.
	10 =	$\frac{N}{10,000}$	solution of alkali.
	11 =	$\frac{N}{1000}$	solution of alkali.
	12 =	$\frac{N}{100}$	solution of alkali.
	13 =	$\frac{N}{10}$	solution of alkali.
	14 =	$\frac{N}{1}$	solution of alkali.

This will emphasize the very slight degree of alkalinity of the blood when we remember that its $_pH$ lies between 7 and 8.

The normal findings of 7.8 to 8 are quickly contrasted with the findings at 7.3 or 7.4 which are found in some cases of extreme acidosis.

(*b*) *The other method, that of Van Slyke*, is based upon the fact that the amount of CO_2 which can be fixed or bound by the plasma is a measure of the reserve alkalinity, and gives us another means of measuring its depletion if there be any.

Freshly drawn venous blood is mixed with a small quantity of

potassium oxalate to prevent clotting, then quickly centrifuged, and the plasma separated. Alveolar air, obtained by forcing air out of the lung, without a deep inspiration preceding, is passed through this plasma while in a separatory funnel, the plasma being shaken around until complete saturation is assured; following which 1 c.c. is measured into the cleverly designed apparatus of Van Slyke and the CO_2 displaced by the addition of acid, the whole being done over mercury.

Normally, 1 c.c. of plasma from an adult can combine with 0.54 c.c. to 0.77 of CO_2. but where the alkali reserve is depleted in acidosis, figures much lower are obtained. In every case in which I have found less than 0.25 c.c. death has followed shortly. The method has been of great value in following diabetics and nephritics, and also where a grave surgical operation—*e. g.*, prostatectomy under general anesthesia—has been contemplated. In some cases local anesthesia has been substituted with gratifying results when the CO_2 figures were so low as to render ether anesthesia inadvisable.

Methods Applicable to Urine.—The recognition of the ketonic acids in the urine has been for many years made use of in detecting acidosis, but as a means of measuring its degree; and has proved inadequate and disappointing, primarily because of the fact that the amount of acid excreted is no measure of the alkali poverty. However, a clinical quantitative method for estimating these may be of some help, and a method of Van Slyke, as yet unpublished, giving a comparatively quick gravimetric determination is anticipated with pleasure.

As I follow the question of acidosis closely the importance of the question of the poverty of the bases impresses itself more and more. The variation in the excretion of NH_3 has been of immense value in following a case, such as diabetes, but now we are not satisfied with that alone, but look to the amounts of Ca and Mg as well. In acidosis accompanying diabetes these have been increased out of all proportion to the other bases. Surely if the proportions as well as the quantities of these bases are disturbed, one can learn that the alkali treatment of acidosis does not consist in giving large doses of sodium bicarbonate alone. It may

well explain, too, why some prominent clinicians have found prepared chalk of much value when administered with the bicarbonate, and others have learned to prefer magnesium salts for cathartics.

Clinical Applications.—This patient is a woman of sixty years, a Russian Jewess, who has been under observation for four years. When I first saw her she had a nephritis, a hypertrophied heart showing a double mitral murmur, and a moderate edema of the lower extremities, with some edema about the eyes and the hands. Soon after she began to improve, the edema lessened, and the appetite returned. I found sugar in the urine, and this has been noted repeatedly. With a change in diet she lost weight and gained strength, the amount of urine increased, the albumin lessened, and sugar was absent when carbohydrates were moderately restricted. The blood-pressure became higher until a systolic pressure of 185, with a diastolic of 130, was frequently found.

One year ago there occurred an embolism of the left lung. Since then she has been fairly comfortable, resting a portion of each day in bed, paying strict attention to diet and elimination. At times she has done a small portion of the housework. For the past week she has noted marked dyspnea, even without exertion, a headache, diffuse and most marked in the afternoon, a complete anorexia, and extreme restlessness. Day before yesterday she vomited, after which she took an ounce of milk of magnesia, and felt some relief after a free bowel movement. I saw her that evening for the first time in months.

You will note the rapid respiration, the aromatic odor of the breath, which can be accurately described as fruity with an admixture of fetor, the dry tongue with glazed tip and margins, the inability to follow any one subject of conversation without a lapse, and the tendency to doze the moment she is not disturbed. Her skin is dry and there is moderate edema of the legs and feet. The temperature is 99° F., the pulse 102, strong, and once in 30 to 40 beats there is an extrasystole. The systolic blood-pressure is 224, with a diastolic pressure of 120; her respirations are 40 per minute and are rather deep.

She passed 640 c.c. of urine yesterday, acid (having a $_{P}H$ of 5.8) of 1023 specific gravity, with albumin, acetone, and diacetic acid. No sugar was found. The amount of NH_3 in twenty-four hours was 1.4 gm.

The amount of CO_2 in the alveolar air was estimated, according to Marriott, as 25 mm.; with the Fridericia apparatus, 3.2 per cent.—both figures about 40 per cent. below the average amounts normally.

The R_PH of her serum was 7.4, a decided lowering when compared with the 7.8, and the CO_2 combining power was 0.33, usually 0.54 or more.

These findings confirm the presence of an acidosis of more than moderate degree. While she is under observation we will follow the trend of the alkali poverty by daily estimation of the CO_2 in the alveolar air, and when twenty-four-hour urine can be obtained by the amount of NH_3 passed. The R_PH of the serum and CO_2 combining power of the plasma will be estimated daily, and even twice daily if the situation becomes critical.

Briefly outlined, the treatment consists of extreme care about quiet and warmth, with a warm pack daily. The diet so far has consisted of 1000 c.c. of raw milk and 50 grams of dextromaltose; the total fluid for the day 2200 c.c. Digitalis is given to support the heart, and diuretin administered both for its diuretic action and because it has a marked influence upon the cardiac apparatus. The bowels are kept open freely, using milk of magnesium daily; the alkali treatment so far has consisted in the administration of 1.5 grams of prepared chalk with 4 grams of sodium bicarbonate in salol-coated capsules at six-hour intervals, thus avoiding their destruction in the stomach and yet furnishing a store in the bowel for the body needs.

Subsequent History.—Four weeks have elapsed since the above findings were recorded. The patient is again at home still confined to her bed, sleeps poorly, but is comfortable during the day. She has no difficulty in taking food, but finds the restrictions irksome. Her temperature is normal, respiration 24, pulse 88, systolic blood-pressure 174, diastolic pressure 110. The urine on last examination measured 1860 c.c. in twenty-four

hours, having a specific gravity of 1027 with 42 grams of dextrose and 0.7 gm. NH_3, a trace of albumin, no acetone or diacetic acid. Hyaline and granular casts were present; the $_PH$ was 6.2. The alveolar air now shows a CO_2 tension of 35 mm. of mercury (Marriott) equivalent to 4.8 per cent. The serum has an R_PH of 7.7 and the CO_2 combining power of the plasma amounts to 0.57 c.c.; all figures showing a return to normal limits comparable to the change in the physical condition which is practically the same as it was previous to the attack.

The diet includes 50 grams of stale white bread with restricted amounts (100 to 200 grams) of vegetables containing 5 to 10 per cent. carbohydrate, all of which are doubly boiled when it is possible. One fast day a week is followed by a day during which nothing but 1500 c.c. of milk is given. Digitalis is still administered, but the alkalies were discontinued after the first four days.

CLINIC OF DR. WALTER W. HAMBURGER

COOK COUNTY HOSPITAL

ACHYLIA GASTRICA

Citation of Typical Cases. Functional and Organic Achylias. Clinical History and Symptoms of 29 Cases. Frequency of Late Pains Simulating Hyperchlorhydria and Gastric Ulcer. Importance of These Cases from the Standpoint of the Mechanism of Gastric Pain. Psychic and Chemical Achylias. Gastric Motility, Gastrogenic Diarrhea, and Gastroptosis in Achylia. Treatment.

I DESIRE to present to you today a group of stomach cases which have been of particular interest to me within the past few months. These cases may be grouped under the general caption *Achylia Gastrica*. They are of special interest from the standpoint of diagnosis, but they have been called to my attention recently because of the similarity of their clinical history to that of chronic gastric and duodenal ulcer; that is to say, they bring up vividly the question of the mechanism or production of gastric pain, a subject which, as many of you know, we have discussed on several different occasions in this clinic.[1] You will remember that it is our belief that the so-called "hunger-pains" in chronic ulcer are caused by sudden and vigorous contractions of the stomach, and are not due to irritation of the ulcer base by the acid content of the gastric juice.

This series of cases of achylia emphasize this belief in so far that many of them, as you will note, give the classical history of

[1] Abdominal Pain, Clinic of Dr. Walter W. Hamburger, Medical Clinics of Chicago, November, 1915; Treatment of Gastric Ulcer, Clinic of Dr. Walter W. Hamburger, Medical Clinics of Chicago, November, 1916.

ulcer or hyperacidity, but, in fact, possess neither ulcer nor hyperacidity.

I shall, therefore, present to you somewhat in detail a number of these cases so that you may judge for yourself, and then later perhaps we can give the results of a series of these cases with some references to the literature regarding others.

The first patient (J. A. W.) I wish to present to you is this young man, thirty-two years of age, a bank clerk by occupation. He entered the hospital the latter part of May, 1916, complaining of:

(1) Epigastric distress.
(2) Vomiting.
(3) Precordial pain.
(4) Constipation.
(5) Loss in weight.

These complaints have been present for eight years, since his twenty-fourth year. He states that the pain occurs fairly regularly two hours after meals; that it is dull in character; that it radiates somewhat to his "heart region" and the "spinal column." He has noticed that almost any food will initiate this discomfort, but the less he eats the quicker the pain occurs. Soda will relieve the pain to some extent, also food temporarily. He has not been awakened at night with pain. The vomiting has occurred periodically, mostly on an "empty stomach," four or five hours after meals. The precordial pain of which he complains is simply the discomfort resulting from the radiation of the original epigastric pain. He has been constipated somewhat all his life. His appetite is good, but he has limited his food intake because of the fear of eating and the fear of producing his gastric discomfort. He has lost 40 pounds in weight during the past six months.

On *examination* he reveals an exceedingly thin, emaciated, pale young man. There is slight general adenopathy of the superficial lymph-glands. His eyes, mouth, teeth, tonsils, thyroid are all negative. His chest reveals a slight hyperresonant note on percussion. His cardiac outlines are within normal boundaries. There are no murmurs. His pulse is regular. His temperature is somewhat subnormal, ranging from 97.8° to 98.4° F.

His abdomen is narrow, but soft and relaxed. There are no tender areas. The liver in the right hypochondrium may be indistinctly palpated and there is slightly increased resistance, although not a definite palpable mass to the left of the umbilicus.

His blood, urine, and blood-pressure show no untoward findings.

A fractional meal reveals the following:

8.00 o'clock Fractional Meal: 300 gm. tea, 10 gm. lactose.

Time.	Free acid.	Total acid.	Lactose.	Bile.	Blood.
8.30 o'clock	0	14	plus	0	0
9.00 o'clock	0	20	plus	0	0
10.00 o'clock	0	8	0	0	0
9.30 o'clock	0	14	0	0	0
10.30 o'clock	0	18	0	0	0
11.00 o'clock	0	12	0	0	0

You will note the total absence of free acid for a period of three hours after the test-breakfast of 300 grams of tea and 10 grams of lactose, a total acidity varying from 8 to 20. In other words, there is a complete absence of free hydrochloric acid, or, as commonly called, an achylia gastrica.[1]

x-Ray and *fluoroscopic examination* reveal no defects in the stomach or duodenum. The greater curvature and pylorus were slightly below the line of the iliac crest, the duodenum was in normal position. The stomach emptied itself completely in six hours.

Repeated stool examinations were negative for occult blood.

Diagnosis.—In view of the fluoroscopic and laboratory findings, we made a diagnosis of an achylia gastrica simplex and general visceroptosis.

Therapy.—The patient was put to bed, the foot of the bed elevated 6 inches, and he was given a general ward diet, to which additional fat and carbohydrate was added to give it a high caloric value. He was allowed raw fruit and raw vegetables and coarse food of all description. It was a little difficult for a few days to

[1] True achylia gastrica implies the absence of both free hydrochloric acid and pepsin, but as commonly used in clinical parlance it is applied to this type of case in which free hydrochloric acid is entirely absent, irrespective of the presence or absence of ferments.

get him to take an adequate amount of milk and cream and a sufficient number of eggs because of his insistance that eggs and milk did not agree with him. However, with the aid of the dietitian and the floor nurses sufficient eggs and cream were added to his diet in the form of egg chocolate, milk shakes, egg-nogs, and egg-and-cream desserts, so that at the end of a week he was taking 6 eggs a day and 1 quart of milk and $1\frac{1}{2}$ pints of cream. At the end of three weeks he had gained 11 pounds. At this time he was discharged from the hospital. He has reported back at intervals since then, and during the following three months he not only regained his entire 40 pounds but added 10 to 15 more.

With the assurance that he had neither ulcer nor cancer, and that he had been taking large quantities of milk and eggs, partly without his knowledge, the patient was fully satisfied with his "cure."

Before coming into the hospital he had been under observation at another institution, where he had been advised that he had a duodenal ulcer and that only an operation would cure him. Were one to base an opinion purely on his *history*, I think all of you would agree that this patient had in all likelihood a chronic ulcer of eight years' standing.

In retrospect then: This patient presents an example of a man suffering with a chronic abdominal complaint of eight years' duration, consisting of pain two hours after eating, vomiting, and loss in weight, but in whom no direct evidence pointing to chronic ulcer could be detected, and who made a rapid and uneventful recovery on a full nutritious diet with rest in bed.

The second patient (H. J.), a young woman, twenty-nine years of age, presents a somewhat similar history. Her complaints, lasting for eight or ten years, consisted of:

(1) Gnawing pain in the stomach.

(2) Extreme hunger.

(3) Pressure and fulness after eating.

The pain which she describes as being gnawing in character does not bear as constant relation to her meals as in the first patient, although it occurs at times when her stomach is empty;

that is, late after meals. In addition, she complains of this rather constant hunger or bulimia, and feels that she could eat almost at any time, even shortly after a full meal.

She also complains of weakness on slight exertion, of lack of endurance, and of inability to gain in weight, although she has not lost markedly.

Her bowels move daily without assistance.

Physical examination reveals a slender, gracile young woman, suggesting somewhat the stature of a tuberculous patient.

Physical examination of her organs is negative.

A fractional test-meal shows the following:

Time.	Free acid.	Total acid.	Lactose.	Bile.	Blood.
8.30	0	14	plus	0	0
9.00	0	8	plus	0	0
9.30	0	12	plus	0	0
10.00	0	8	plus	0	0
10.30	0	16	0	0	0
11.00	0	10	0	0	0

x-Ray examination shows an atonic stomach with a considerable hypersecretion. The greater curvature is one finger below the umbilicus. Motility, six hours.

A *diagnosis* similar to the first patient was made in this patient; that is, achylia gastrica and enteroptosis. She was given the same treatment, rest in bed and overfeeding. She gained 12 pounds in weight in five weeks, with a parallel improvement in her general condition.

Before leaving the hospital she was given a second fractional test-meal with the following results:

Time.	Free acid.	Total acid.	Lactose.	Bile.	Blood.
8.30	0	7	plus	plus	0
9.00	0	10	plus	plus	0
9.30	6	12	plus	plus	0
10.00	10	18	plus	plus	0
10.30	4	12	plus	plus	0

In other words, one and a half hours after giving the fractional meal she showed a free acidity of 6, which at the end of two hours went up to 10. In other words, the second hour of her test-meals showed in all aspirations the presence of free acid

in contrast to the complete absence of free acid in all aspirations before treatment was commenced. I shall refer to this later in our discussion of the fractional meal in achylia gastrica.

The subsequent history of this patient, however, was not quite as satisfactory as the first one. After discharge from the hospital and taking up her usual duties she again complained of some gnawing sensation after meals. This, however, was promptly remedied by the application of a properly fitted Curtis abdominal support.

In summary, then, these two cases demonstrate examples not only of achylia gastrica but of an associated gastroptosis. While achylia and visceroptosis or gastroptosis are frequently associated, they are not necessarily so, as either may occur separately. In our experience they are very frequently found in the same patient, but not always so, as may be seen from the following case:

(A. F.): Hat maker, forty-four years of age, complains of vomiting two hours after meals and constipation. He has had no loss in weight, no pain, no nausea or fulness after meals.

Three Ewald test-meals show constantly complete absence of free acid; total acid ranging from 6 to 15. The greater curvature of the stomach in the upright position was three fingers above the crest. Stomach emptied itself completely in four hours. Diagnosis: Achylia gastrica simplex.

Definition of Achylia Gastrica.—What do we mean by achylia gastrica? In modern clinical parlance most clinicians mean cases of the type we have just shown you; that is, patients in whom repeated test-meals show total absence of free acid and low total acidity. The name *Achylia Gastrica* was first introduced into gastro-enterologic nomenclature by Einhorn in 1892, who described several patients in whom both free hydrochloric acid and pepsin were absent, but in whom pathologic changes in the gastric mucosa were absent or at least of minor importance. In other words, Einhorn believed that he had described a new clinical entity, a primary or *functional* abnormality of the gastric secretion, and not a secondary organic atrophy of the gastric glands, secondary to chronic gastritis.

Quincke (1871) and Fenwick (1877) had, it is true, previously described cases of atrophy of the gastric mucosa, an *organic* achylia; and Levy, Ewald, Rosenheim, and Jaworski had described some of the clinical symptoms of the disease. In 1897 Martius and Lubarsch published their classical monograph on achylia, which presented the complete clinical and pathologic material of this disease.

The clinicians and investigators interested in this disease, as in many instances in medicine, are not agreed as to its ultimate pathology, and they may be grouped into two divisions—those who believe in an *organic* basis for the achylia and those who hold for a *functional.*

Knud Faber, of Copenhagen, is perhaps the most ardent advocate of the organic achylias, believing that all true achylias are resultants of a preceding chronic gastritis.

Lange, Elsner, and Rosenheim subscribe to this same belief. Others, however, such as Einhorn, Martius, Albu, Zweig, Rudolph Schmidt, and Boas, believe that achylia does not *always* mean an atrophy of the gastric mucosa, while Disque believes that the functional achylia is much more frequent than the organic.

My own experience, while extremely limited in comparison with that of the men just quoted, likewise leans to the greater frequency of the functional variety.

In the patient H. J., the second patient presented to you, you will remember that there was a beginning return of secretion in the second hour of digestion after five weeks of treatment. Naturally, this could not have occurred had there been a complete atrophy of the gastric mucosa. I recall in this connection seeing a young man several years ago who came to me because of frequent vomiting spells shortly after eating. At the time of the onset of this complaint he was suffering from a severe mental strain incident to an unhappy love affair. Repeated test-meals showed complete absence of free acid. With a cessation, however, of his mental unhappiness his vomiting promptly disappeared. Test-meal examination some months later showed a normal gastric juice.

Clinical History and Symptomatology.—The point, however, that I wish to make today is the *clinical history* of many of these patients, and to emphasize this more strongly I have collected statistics on 29 cases of achylia from the histories of patients in the Cook County Hospital:

Thirteen, or very nearly half, of these patients complained of *gastric pain* coming on from *one-half* to *one and one-half* to *two* hours after meals.

Four patients timed their distress from *two* to *three* hours after meals.

Two patients, from *four* to *five* hours after meals.

Seven patients complained of irregular distress; that is, distress which either did not amount to true pain, either had no relation or a bizarre relation to meals, or was colicky and present in the lower abdomen.

Seven patients in this group complained of vomiting; 3, of nausea; 2, of anorexia; 3, of diarrhea; 2, of constipation.

Of most significance, I believe, is the first-mentioned fact, that is, that 19 of these 29 patients, or practically two-thirds, complained of *definite epigastric pain coming on from one-half hour to four and one-half hours after meals.* The majority of them, nearly one-half the total number, complained of pain within the two-hour period after meals.

You will agree, I am sure, that such pain is often said to be typical of gastric or duodenal ulcer. But in the analysis of these cases and in their subsequent improvement there was at no time positive evidence of an ulcerated condition. What, then, is the explanation of their "hunger-pain"? This, as we have said, we believe is due to sudden increase in tonus contractions of the stomach. Cases of this type explain (1) the *absence* of ulcer in patients subjected to laparotomy on the ulcer history alone; (2) the frequent prompt *cure* of "ulcer" by simple rest in bed and wholesome food.

To my mind they are perhaps the most instructive and enlightening group of all cases in gastro-intestinal disease. Einhorn, in 1901, published 6 cases of achylia gastrica simulating hyperchlorhydria (Achylia Gastrica Hyperchlorhydrie Vortaüschend).

These 6 cases with hyperchlorhydria symptoms presented examples of epigastric pain occurring from one-half to three hours after meals, some of them associated with diarrhea and vertigo. Many of them were treated as hyperchlorhydria without results. After the correct diagnosis had been made there was a rapid improvement.

To my mind, cases of this type, including this series which I have gathered, present incontrovertible evidence in favor of the newer theory or mechanism of "hunger-pain," and strongly refute the older theory of the irritating hyperacidity of the juice. That this theory is gaining wide recognition may be evidenced by the fact that in the recent publication of "Monographic Medicine," Vol. I, page 172, Hewlitt has the following to say:

> "Providing there is no disease of the peritoneum, gastric pains appear to be caused mainly or solely by the muscular contraction of walls of the stomach or by their mechanical distention. In other words, gastric pain is caused by increased tension of the muscle wall. . . .
>
> "It seems evident, therefore, that one and probably the most important cause of gastric pain is an increased tension of the muscular elements of the stomach wall."

However, I do not wish to give you the impression that pain is the striking complaint of these patients. While in this particular series the majority of them complained of some pain, several of them complained only of vomiting, anorexia, weakness, etc. As Disque has emphasized, many of these patients have no gastric complaint whatsoever, the achylia only being found on the passage of a stomach-tube.

Rudolph Schmidt has recently (1912) pictured the clinical findings of what he called *essential* or *constitutional achylia*. He emphasizes a number of points which are of frequent occurrence in all these patients. First, many of these essential achylias are young, weak, nervous, and of asthenic appearance, with somewhat the general appearance of tuberculous patients.

Second, they may often show some congenital malformation, such as congenital diastasis of the recti, anomalies of the colon, hernia, etc.

Third, they may show changes in their reflexes, markedly increased abdominal reflexes, increased hyperesthesia in the right lower quadrant of the abdomen.

Fourth, they often have a cramped condition of hands and feet, reminding one of tetany patients.

Fifth, nausea and vomiting are frequent, often immediately after eating, even, at times, containing blood.

Sixth, they complain of acid diarrheas, the so-called gastrogenic diarrheas.

Seventh, they may have a disturbance of taste, such as sour or bitter taste; small vesicles on the tongue, similar to mouth conditions of pernicious anemia.

Eighth, they often have a marked bradycardia or a variable pulse, often with low blood-pressure.

Ninth, many have an orthostatic albuminuria.

In Schmidt's experience the triad of achylia, bradycardia, and a predisposition to tuberculosis has been marked.

Psychic and Chemical Achylias.—The most recent work on achylia with which I am acquainted has been published by Rehfuss in the "American Journal of the Medical Sciences," 1915, Vol. CI, p. 72. Rehfuss divides the achylias into psychic and chemical achylias, the psychic or nervous achylia being present in the first hour, the chemical or secretory achylia in the second. In his cases he found both psychic and chemical achylia, or either one or the other alone. In his experience the commonest form was total achylia occurring in both phases. He cites 7 cases, 2 of which show no gastric complaint whatsoever; 2 cases with discomfort and distress occurring regularly after meals; 1 case beginning with nausea and vomiting, and 2 cases beginning with epigastric pain. He found the administration of parathyroid extract, following a suggestion of Carlson's, was of value.

Gastric Motility in Achylia Gastrica.—There are a number of other points of importance in connection with the subject of achylia that I wish to discuss with you briefly. One is the question of *stomach motility*. As you know, there are only two important functions of the stomach—one secretory, the other

motor. In achylia gastrica the secretory function is markedly impaired; that is to say, the ability of the gastric glands to secrete normal gastric juice containing free hydrochloric acid is lost. What about the motility? In what way is motility affected? It has been the general belief for many years that in achylia gastrica the motility of the stomach is very much increased, the stomach emptying itself much quicker than in a normal individual. For instance, Boas describes it as a hypermotility similar to the hypermotility found in incontinence of the pylorus, gastric neurosis, chronic gastritis, bulimia, and so on. However, in the cases which I have had the opportunity of studying, both by motor-meal and *x*-ray, the motility has been exceedingly variable, sometimes, it is true, increased, but just as frequently normal or even delayed. In two of the cases shown here this morning you will remember their emptying time with the bismuth meal was six hours, the upper limit of normal. Elsner (1904) comes to the same conclusion, finding that the emptying time in achylia gastrica may be normal, less than normal, or greater than normal. He believes that the condition of achylia *per se* has no constant effect upon gastric motility, stating further that the belief that a diminution of gastric acid is compensated for by increase in gastric motility is erroneous, at least in a large series of cases. In this connection some recent work of Spencer, Meyer, Rehfuss and Hawk ("American Journal of Physiology," February, 1916), and Morse ("American Journal of Physiology," October, 1916) is of interest. They discuss Cannon's work on the acid control of the pylorus, and find that the physiologic evidence that the presence of an acid reaction in the stomach is essential and hastens gastric discharge is not entirely concordant with clinical observations, particularly in cases of achylia gastrica where the normal rate of discharge is often unimpaired. They find in general from experimental work on dogs that the presence of acid in the stomach has been observed to retard rather than hasten gastric discharge, and believe that the acidity of gastric contents has no function in either producing or hastening gastric discharge, but that the discharge seems to be controlled by the irritability or non-irritability of gastric contents on the duodenal mucosa.

Gastrogenic Diarrhea.—Another question of interest is the problem of the gastrogenic diarrheas already referred to. It has been the belief for some time that the cause of diarrhea in achylia gastrica is the failure of pancreatic, biliary, and intestinal digestion to entirely supplement the absence of digestion in the stomach; that is, the undigested food material entering the small bowel sets up an enteritis, and finally an enterocolitis, and therefore a diarrhea. However, in my own experience the occurrence of diarrhea has been exceptional; a normal or sluggish bowel function the rule. It is possible that in the same way that gastric motility was found normal or delayed, intestinal motility is likewise normal or delayed. At all events, the frequency of gastrogenic diarrheas in this series has not been prominent.

Gastroptosis in Achylia.—The third and last point I wish to mention is the association of gastroptosis or visceroptosis with the achylias. That they frequently occur has already been discussed. In Rovsing's series of 300 cases of gastroptosis a normal acid content was the rule, and in the second largest percentage an achylia.

In my own experience the frequent association of achylia and gastroptosis has been striking.

Treatment.—It is obvious that adequate treatment cannot be instituted before an accurate diagnosis is made, and an accurate diagnosis cannot be made until a test-meal is given and the actual achylia demonstrated by aspiration. Achylia gastrica is one gastric complex in which no other method of diagnosis is dependable. While indirect evidence of an achylia may be suggested by the finding of undigested food remnants in the stool, an absolute diagnosis can only be made by test-meal investigation. After a positive diagnosis has been made one searches for causes of the achylia and attempts to rule out the secondary or organic achylias, a resultant of chronic gastritis, carcinoma of the stomach, pernicious anemia, and other toxic agents. These various diseases having been ruled out, and if one has diagnosed an essential or constitutional achylia, proper treatment can be instituted. This is well summed up in Schmidt's excellent article.

"Treatment," Schmidt says, "is to be directed less to the

stomach than to the gastro-intestinal tract; less to the gastro-intestinal tract than to the general organism. One should think of rest cures, of sojourns in the mountains or by the sea; individualize your patients; test their alimentary tolerance and intolerance; no diet schemes; treat your achylias less as stomach specialists, but more as general practitioners."

In my own experience most of these thin, undernourished patients should be put to rest in bed, at least for a few weeks. Raising the foot of the bed is of value, provided there is an associated visceroptosis. If there is no tendency to diarrhea secondary to their poor gastric function, they should be put at once on a full, nutritious diet of wholesome vegetables, fruits, bread and butter, milk and cream, eggs, simple, well-prepared meats, fowl, and fish. If they are underfed, the caloric value of their diet may be raised, with additions of fat and carbohydrate in the form of cream, butter, cheese, cereals, rice, etc.

If there is evidence of a secondary colon involvement of the nature of a colitis, spastic colitis, or diarrhea, their vegetables and fruits should be thoroughly cooked and strained through fine sieves; their meats should be well cooked and chopped or minced. All rough, cellulose-containing food should be eliminated from the diet.

In the way of medication, 10, 15, or 20 drops of diluted hydrochloric acid added to a full glass of water, to be taken before, during, and after meals, is apparently of some value. It is often surprising how the hyperacidity complaints are relieved by the addition of more acid!

Other patients, however, gain more relief from the paradoxic administration of weak alkalies, such as sodium bicarbonate, sodium citrate, or bismuth subcarbonate.

One may, if he is venturesome, attempt the administration of calcium chlorid or parathyroid extract, suggested by the experimental work of Keeton.

After discharge from the hospital, corrective exercises, out-of-door sports, frequent periods of rest, massage, hydrotherapy, etc., may all be of value.

Measures to improve the general asthenic condition of the

patient may be in order. Psychotherapy, providing there is definite evidence of mental strain, is sometimes of great benefit. Assurance that their trouble is nervous in origin and will improve in all probability with the cessation of nervous and mental hardships may be helpful. I have in mind particularly a young woman whom I have just had under observation. She is the superintendent of a large home for friendless children. She is intensely interested and active in her work, constantly initiating and supervising new activities in the institution over which she has charge. Her complaint is constant hunger and weakness. This summer, before starting on her vacation at a time when she was "all in," she was 12 pounds underweight and had no free acid in any of her stomach aspirations. Three weeks in the country, with rest, recuperation, fresh air, and good food, restored one-half of her lost pounds, and analysis this week of her gastric contents showed a beginning return of the first-hour acid, the second-hour phase still being absent.

Just one word more. Mention has been made of the necessity of ruling out the large group of conditions which at times is responsible for the production of an achylia. This group is legion, but nevertheless must be considered in the differential diagnosis of achylìa gastrica simplex. Following the classification of Riegel, they may be considered as follows:

I. *Conditions causing direct damage (organic) to gastric secreting glands, productive of anadenia:*

Severe acute and chronic gastritis.

Carcinoma of the stomach.

Advanced carcinoma of distant organs.

Amyloid degeneration of gastric mucosa secondary to tuberculosis, syphilis, or chronic suppurative process in body.

Pellagra.

Sprue.

Pernicious anemia.

Severe secondary anemias—chlorosis.

II. *Conditions causing temporary or permanent functional inhibition of secretory nerves (vagus):*

Achylia gastrica simplex.

III. *Conditions causing disturbance in intermediate chlorid metabolism resulting in deficiency of hydrochloric acid.*

Cardiac disease—vascular heart lesions; myocardial disease.

Chronic interstitial nephritis.

Liver and gall-bladder disease—cirrhosis; cholecystitis; cholelithiasis.

Diabetes.

Gout.

Wide-spread atherosclerosis.

Cachexias and severe nutritional disturbances.

In this connection, one remembers that chronic, long-standing gastric and duodenal ulcer may be associated with a relative or complete achylia. E. S. Crispin ("Interstate Medical Journal," October, 1916) has most recently again called attention to this, citing "11 cases in the Mayo Clinic of operations proved duodenal ulcer in which the gastric analysis showed an absence of free hydrochloric acid."

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NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

SOME CONSIDERATIONS OF THE PROBLEMS OF PSYCHIATRY

Importance of a Proper Understanding of the "Neurasthenic" State. Analysis of What is Going On in the Patient's Mind. First Symptoms of Mental Disease Often Physical. The Religious Impulse. Early Sexual Impressions.

October 9, 1916.

THE patient to day is a mental case which I want to show you, inasmuch as mental disease is a very large part of the disease of the world.

This woman was brought to Wesley Hospital on September 6th. She is single, aged fifty-seven years. She came in complaining of purely physical symptoms, as is so frequently the case—vomiting, cramps, constipation, and soreness in the abdomen. She has had a "weak" stomach for three or four years and has been vomiting for the past year. She is first nauseated, with much belching, then gradually cramps come on, and finally the vomiting occurs. In the last week the cramps have been much worse. Last Friday, on going out for a walk, her head began to whirl and she fell to the ground; about five minutes afterward she became unconscious (?) and remained so until the following morning at 4 o'clock. After 4 o'clock she had some abdominal distress, but after a while fell asleep. When she awoke she still had cramps and vomited. This continued through the following day, and the next day she had cramps and fever, but did not vomit. For the past two weeks she has been having injections of warm water for the bowels, and these make her feel better.

For the past eighteen months she has not eaten much meat or "heavy" food of any kind because it would cause her much distress and vomiting. She says she was overcome with stove-gas three or four years ago, and fifteen years ago she was in a trance for three months. The history says "comatose," but that was not the case. Comatose implies a severe physical condition, but a trance is quite a different thing—it is something that a person gets into from a subconscious or conscious state of mind.

She has two sisters in Europe. Her mother died at thirty; the father remarried, and the stepmother would beat her and the other children.

She dates her first gastric distress about fifteen years ago when she had the trance. As the result of lifting a heavy weight she had a severe pain over the epigastrium. This pain was constant for fifteen months. There was no vomiting at that time. Since then she is seized with the same sort of pain whenever she lifts a heavy weight. She has never vomited blood, but the vomitus contains a large quantity of bile. She is very constipated, sometimes going eight days without a bowel movement. She has never been jaundiced.

Further inquiry elicited the fact that she has had palpitation attacks ever since eighteen or nineteen. Attacks of palpitation are the commonest of symptoms of all mental states because so often the mental state depends on a lack of self-confidence—a feeling of apprehension—and if you translate the mental state into physical symptoms one of the first will be palpitation. It is a physical descriptive term of fear—"my heart is in my mouth" or "I have a smothering sensation." Another feeling of fear is the sinking in the pit of the stomach and another a feeling of weakness behind the knees. In the olden times they spoke of a man's knees being loosened; in modern times we speak of "cold feet." When palpitation attacks occur, perhaps for a perfectly good reason, persons who are apprehensive do not realize that their physical symptoms are due to a mental state, with the consequence that they say they have heart disease or trouble with their stomach or their lungs.

The relationship between these apprehensive attacks and

states of physical disease is a twofold one: either the attacks may be due to a mental cause, an emotion with consequent physical reactions, or there may be some intoxication process which produces physical symptoms such as palpitation, smothering sensations, etc. In the latter case where there is a real physical basis which is sufficiently hidden so as not to be readily appreciated by the sufferer, the symptom, being one of fear, seems to bring about the emotional state of fear. (Patient is brought into the clinic.)

Miss M. did not want to talk to you, but I want her to tell you what is the matter.

DR. HAMILL (to Patient): What seems to be the matter with your stomach, Miss M.?

PATIENT: It hurts me so across here, and I feel so weak and the cramps come. People bother me so.

DR. HAMILL: Has anyone done anything to you?

PATIENT: I don't know; somebody is always talking. Sometimes I am stronger, and then I don't mind so much, and other times I can't stand the remarks people make, and that makes me sick to my stomach, and my stomach is sore and the blankets press too much on my stomach. I can't stand the weight of them.

DR. HAMILL: Why should people say these things about you?

PATIENT: I don't know whether they are jealous or not, but I guess just for meanness.

DR. HAMILL: What was the matter at the time you had the trouble here in the hospital when it seemed as if a snake bit you?

PATIENT: The snake seemed like a wicked enemy, a woman or a man, or a poor thing.

DR. HAMILL: Do you think these cramps have been caused by the poison that seems to circulate in your body when people talk about you?

PATIENT: It hurts in my side, and I get cramps, and the poison goes all through me.

DR. HAMILL: What about the trance that you had fifteen years ago, was it caused by the same kind of feeling?

PATIENT (indignantly): Not at all, that had nothing to do with it.

DR. HAMILL: Have you any idea what the trance was due to?

PATIENT: I know people have trances.

DR. HAMILL: But there is always some cause for it. Do you think God did it to you to make you understand his power?

PATIENT: I think so; I was always strong and well.

DR. HAMILL: How long have you been in the hospital?

PATIENT: Five weeks next Wednesday.

DR. HAMILL: What day is today?

PATIENT: I think it is the 7th or 8th of October.

DR. HAMILL: What kind of a place do you think this is?

PATIENT: I have never been here before and have never talked before people, but I think this is a clinic.

DR. HAMILL: You could have preached to people, couldn't you?

PATIENT: Oh yes, I could have, but I never did.

(Doctor sends patient from the room.)

DR. HAMILL: You can see that this woman knows where she is and has an approximate idea of the date. She is perfectly conscious of her surroundings, but she has these ideas which she is not willing to state in this crowd as she does when I am with her alone. It is perfectly apparent at times that she imagines all of her physical symptoms are due to the fact that people are mean to her, or that she thinks they have some supernatural power over her.

Her history, as she gave it to me, is more or less like this: She never knew her own mother. When she was thirteen or fourteen years of age her stepmother drove her out to do housework in the neighborhood, telling her that she was old enough to earn her own living. When she was sixteen years old she cut loose from home altogether, and took service in a big house in the neighborhood where there were other servants. One of these was wild, and at one time it came to her just like a voice, saying, "Now you must determine, heaven or hell—now you have to make up your mind; are you going to be good or not?" She was led to tell me that by my asking her when she first felt the religious impulse which she expresses very strongly. She will con-

stantly interrupt her story with an allusion to God, and when I asked her when she first had this religious impulse I found that it was at fifteen to seventeen, as it always is.

Right here I want to speak of the religious impulse because religious ideas play such a large part in many abnormal mental states. From the earliest times men have worshiped something, and the more mysterious and occult the something was, the more influence it had. As children grow up they depend upon their parents for aid and advice. That is perfectly satisfactory so long as they feel that aid and believe that their parents are omnipotent. During that period they have learned various things, and one of those things is that they have not full control over themselves in all matters of passion and temptations. Along about twelve to fifteen children get away from home influences by being sent to school or going to work, and their horizon is sufficiently enlarged to make them realize that their parents are not omnipotent. Then they are looking for help. Most of us have been brought up to go to Sunday school or church and God has been held up as an omnipotent force, and when these temptations threaten to become overwhelming, as they always do around the age of fifteen, this God is appealed to.

So this girl goes out to work, and one night the fellow servant who was spoken of as "wild" takes her by the neck and tries to make her give up. Shortly afterward the voice came to her, asking if she is going to lead a moral life or give way to her impulse and have the fun out of life that it promised. So she had to make the decision, and it is characteristic of this person's mind that it came in the form of a voice that is actually heard. It is not an idea that came into her mind that now she must decide this question, but there is actually the voice. Where you find this you will always find a tendency toward hallucinations later in life. A patient I had who was a kept woman, and who was drinking too much, used to hear people going by the house saying, "A whore lives there." As a matter of fact she was one, but she did not hear people saying it. The alcohol gave her the hallucinosis. In the hospital, with the alcohol taken away, the hallucinosis disappeared. Three and one-half years later she had

a streptococcus infection of the throat and had the same hallucinosis from a different agent.

From fifteen on our patient of today was more and more religious, and, according to her story, she became somewhat isolated from other people. When you dig deeply enough into this you will find the desire to mix too intimately with other people—too intimately in a reprehensible way. In her case there was the perfectly natural sexual impulse of a girl of fifteen. She also had the influence exerting itself on her of the cruel stepmother sending her out from home. You can easily picture to yourself the influence it would have, the thought that "I have nobody to be responsible to, I can do as I please." So there were two rather strong forces at work on her, but she did not give in to them. Why, I am not sure, but I think that fear had a very large part to play, as shown by these apprehensive attacks. When I brought her into the room a little while ago she was shaking like a leaf. In order to further fortify herself she exaggerated the religious impulse as highly as possible. She tells about the trance fifteen years ago, but she is not willing to trust me enough to tell me very much about it. When I asked searching questions about it she was obviously suspicious of me. As she tells me about it, however, she says that it "was perfectly heavenly."

I wish to add the biography of an attack of this sort that a woman wrote out for me last year, where, after going into the Psychopathic Hospital and after having a dream that all her family had been killed, thereby relieving herself of all responsibility for their care, she lay down and slept for the first time in three weeks. She had kept her eyes shut for two weeks and it was "perfectly heavenly." The fact that she was in a place where nothing was expected of her, and that she had disposed of her family to her satisfaction, led her to lie there for two weeks, and that was "perfectly heavenly." Miss M. speaks of her three months' trance in the same way. The woman of last year says that after she had been in bed for a couple of weeks she began to realize things again, and, strangely enough, it was her husband that she first recognized, and then the oldest child, and then the

next oldest, and then the baby. Of course the husband was the one that was the easiest to take care of, and the oldest child—who was about fifteen—was easier to take care of than the younger child. She accepted the least responsibility first, and then the next, and the next. So long as she was needing rest she refused to accept responsibility, and when she was rested she commenced to accept responsibilities, and the least first. So Miss M., after being in a trance for three months, began to accept the world on its own basis. She says it was like being born again.

If you will think of the mythology of the world, the Christian included, you will remember that everyone of the mythical heroes had to be born again. Christ had to be born again; Ulysses had to go down into hell and be born again, and Osiris had to go into the fish, and the fish went out into the Nile, and then into the ocean, and then gave Osiris up, and the same thing is true of Jonah and the whale. So it is with baptism today. They take a child to church and have it sprinkled with a few drops of water on its head and say that it belongs to Christ, and so it is cut off from this wicked world; it is reborn into the brotherhood of Christ.

So this woman goes into a trance, and when she comes out of it her connection with God was very much stronger, just as Christ's was after being born again. Ever since that trance she has been in more or less constant communication with Christ. He tells her what to do and what not to do, and because she is so favored of God other people are mean to her and pick on her. They are jealous of her. There is no position in life that is quite so desirable as to have people envious of us. It doesn't matter whether it is because we have an automobile, or because we can run better or faster, or because we have more money or what—it helps us to feel that we are better than other people. This woman has gotten herself into that state; she is the person that God communicates with directly. In other words, she is almost in the state of the Madonna, and I would not be at all surprised if I found out later that some of these cramps have to do with childbirth—the birth-pains of the Madonna. I have just had a case in an epileptic girl who has had, following her epileptic at-

tacks, a period of delirium, and in this she thinks back to the time of her epileptic period and to the abdominal cramps by which she thinks she is giving birth to Christ. Every woman who is brought up in a strict moral sense is naturally afraid of sexual experience. On the other hand, every woman naturally wants a child. Consequently, this girl fancies herself the Madonna, the one woman who had a child without having a sexual experience, and so is going to have a child. I would not be surprised to see something of this kind in this case. She is a mortal who is in direct communication with God. But she can't accomplish miracles; she feels the same bonds we less fortunate mortals do, she even has to work for a living or starve, and mend her clothes or they will fall off. Consequently, there is some discrepancy between her ideas and the real state of affairs, and who is to blame? These people all around who are talking about her all the time.

The other day a man came in to visit his wife who was in the bed next to her. Suddenly she heard him say, "You old whore, go out and buy some furniture," and this referred to her. I have seen this man and know that he would not say anything of the sort—not even to his wife.

Is this dementia præcox? This woman has earned her living up to the time she is fifty-seven. There is a condition known as the *catatonia of involution* which comes on at the time of the menopause. Her menopause was two years ago; her trance was fifteen years ago. In catatonia persons go into a trance; they lie with their eyes closed; they won't eat, they won't sleep. This woman went through this fifteen years ago. Is that catatonia? Is it dementia præcox that is now in the demented period?

I would like to go into it even more in detail than I have just with this in view, that the ordinary practice of psychiatrists is to say, "This patient has dementia præcox, send her to the insane asylum, we can't do anything for her"; or, "This patient has manic-depressive insanity and will have recurring attacks all her life," satisfying themselves with the text-book name and the prognosis that is hung on that name. No case of dementia præcox is always demented; it is not true that all manic-depressive

insanities have recurring attacks. Dementia paralytica may present one picture at one time and an absolutely different picture at another time.

The striking thing about this patient now in the ward is that she attitudinizes to a great degree. She sits up in bed, and has some sort of a grimace on her face almost all the time in the nature of a self-consciousness, as if she had to show people that she is thinking all the time, as if she did not dare be caught without the expression of thought on her face. When she is asked a question she draws down the corners of her mouth and puckers up her brows, as if to say that she is thinking deeply over her answer to the question. That, of course, is seen in a greater or less degree in all people. None of us are willing to be caught with an absolutely inane look upon our face, and if anyone does catch and tell us of it we do not like it. So this woman, who is abnormally conscious of her relations to the world, has to overdo that sort of thing. There are wards in the insane asylums that are filled with people going through some sort of contortions all the time, all the way from slight grimaces to exaggerated stereotyped movements. Some of them will keep putting their arms up and down, and will perhaps wear a callus on their sides from the constant rubbing, or they will move their legs in a stereotyped way thousands of times a day, or you will see them with their back against the wall bending up and down, up and down for hours at a time every day. You will perhaps remember in Mark Twain's "Yankee at King Arthur's Court" how he had one of these fellows harnessed up to a sewing machine so that he could run it for him. The movements that this woman goes through are the same sort of thing that we see in people who are very crazy, people who are not able to stay out of an asylum at all; but this woman restrains her grimacing and attitudinizing even as she has restrained her conduct, so that she has never had to go to an asylum.

I want to convey to you the idea that there is nothing that is merely accidental or haphazard in the development of a case of insanity. I am certain that if we knew enough we could trace every step in the development of an insanity as we went along,

and we would arrive at a conception of the very crazy without any great variation from the logical course of cause and effect. The case that I told you of, the woman who lay with her eyes closed for three weeks, did not eat, did not recognize people, had involuntaries in her bed, and paid no attention to them, and yet she had good reason for doing as she did. She was tired out, and she overheard her doctor and her husband say that she must have a rest. She felt the truth of this, and yet she had the responsibility of her young children. How was she going to work it? The conflict of her feelings of necessity—that she had to get a rest—and the feeling that she had to take care of her children worked upon her until she went "up in the air," and cried and screamed and prayed to God, and sang hymns, and all that sort of thing, and then in the midst of it came this beautiful dream that all her family had been wiped out, whereupon she lay down and closed her eyes for three weeks, which is not such an illogical thing to do after all. If we could *believe* what we *think* we might all do the same thing, but fortunately there is something underlying our thought so that when we do have an incorrect thought we do not act upon it, but that woman after her dream just laid down and took the rest she had to have in order to get her health back.

So it is with attitudinizing. If you are talking to a person you do not just blankly look off into the distance, but you show some sign that you are conversing with this person. Similarly, as you go along the street you reflect in your appearance that you are conscious of the things going on around you. This patient simply overdoes things, and yet because of her grimacing we are very apt to make the diagnosis of dementia præcox. Just why we must overreact to our environment is not so far under the power of our understanding. A person who is naturally very diffident very often overreacts and is called a snob. His diffidence would lead him to bow down to everybody, but he cannot do that, so he reacts too much on the other side and is called a snob. It is not difficult to see that this is the same sort of overreaction to one's environment that you see in Miss M. If we appreciate this fact, then we can see that she is better off away from an environment which demands such reaction. So this woman likes

to stay in bed, she spends most of her time in bed, and has even requested that she be moved into the bed at the end of the room next the window, so that she will have people only on one side of her, and in this way she gets away from the environment that demands overreaction. That amounts to withdrawal from the world. It is that thought that leads nuns and monks to withdraw from the world. We do not call them crazy, but it is the same type of overreaction. They have the same environment all the time and do not have to meet the demands that a changing environment puts on them. That is called a withdrawal from reality.

The mind of the neurotic finds the world and its responsibilities too hard. I think you can say without fear of controversion that there are people all the way from the most social being to the idiot without skipping a single step from sanity to insanity. There are people whose social obligations have no deterent influence on them whatsoever. At the other end of the scale is the idot who acknowledges no responsibility, and I think between the two extremes there is simply a sloping plane without a break in it anywhere. The idea that an insane person is a different kind of animal is an impression that one must get out of one's mind if one is going to understand mental diseases at all.

The neurotic belongs in between the extremes. Take the hysteric woman over at the County Hospital who hiccups by the hour. There is no need of it, it is not an anatomic thing at all —it is obviously a faked hiccup, and yet she keeps it up by the hour. I recently observed a girl of seventeen who was supposed to have epilepsy and in her attacks she had convulsions. During one attack she fell to the floor and cut her head. She developed a paralysis of the arm and held her arm in this fashion (demonstrating) close to her side for two weeks. Her convulsions came on after her father hit her on the head for flirting with a boy in a picture show. I could throw her into a convulsion any time by tapping her on the head with my knuckles. She would get just as blue as any epileptic I ever saw, she would hold her breath and get cyanotic, and then she would begin to twitch and jerk all over. I told her before she had one of the fits that after the

next fit her arm would be all right. While she was in the fit I held her arm out and straightened it, and during her convulsion I saw to it that that arm moved about as the other one did. Then I spoke sharply to her, she came out of her fit, saw the "paralyzed" arm moving like the other one and she didn't hold it fixed any more. It certainly was a crazy idea to hold her arm doubled up; it certainly could not have been comfortable.

The term "neurasthenia" covers all sorts of stages between the normal and abnormal. Many of these neurasthenics are physically sick; they have anemia, constipation, trouble with the stomach, or they have syphilis. The physical disease makes them feel an inequality if they are of the type of mentality who can't see the truth of what is going on, just as this woman, who, in order to decide whether she should be moral or immoral, had to hear the voice say, "You must decide between heaven and hell." If they are sick they feel that the world and society demand certain things of them, and they have to make the most of their excuses for not filling the expectations. They are able, like Miss M., to take advantage of the physical disease to make it an excuse for a retreat from the conflict of competition. They must make the most of this physical disease. So you hear these people complaining bitterly of their stomach symptoms, and yet a minute later if a little excitement comes along their stomach symptoms apparently do not exist; or, if you give them some simple remedy and they believe sufficiently in you, these stomach symptoms disappear. It is a foolish way to behave—to exaggerate one's feelings of illness—yet we don't call these people insane. One might call the neurasthenic the first step and the hysteric the second step toward insanity. We all have had moments when we have not shown all the will power and decision that we might have.

How does this help us in the consideration of mental diseases? If we can get a person in the early stages of neurasthenia, or a person who is not able to understand themselves, and get an understanding of what is really going on in their minds, and then make them understand what consciousness is doing to them in giving them a crooked point of view; then if they have not some anatomic disease of the brain, such as syphilis, we have a chance to

help straighten these people out. Consequently, the understanding of mental diseases is of tremendous importance to any man who is practising medicine, and especially an understanding of the neurasthenic. When you think of how many people are simply called neurasthenics and given a rest cure it is astounding.

So I would make a plea that when you have a mental case you try to understand what is going on in the patient's mind, no matter whether it is a hopeless case or not, because these cases are always an education for the medical man, and each case helps to a better understanding of the others.

ACUTE DISSEMINATED MYELITIS AND ACUTE SYPHILITIC MENINGOMYELITIS

Differential Diagnosis of Two Cases of Acute Disease of the Central Nervous System. Optic Nerve Changes. Prognosis and Treatment.

TODAY I wish to show you two cases of acute disease of the central nervous system. Both of the patients have, in a large measure, recovered, and the condition we see today is the result of the action of the acute disease.

The first case is a school-girl, thirteen years old, who entered the County Hospital on May 5th. She complained of pain over the entire abdomen and to a lesser degree in the lower limbs. The pain in the abdomen was of such striking character that the intern in the examining-room made a tentative diagnosis of "general peritonitis." The mother's account of the illness is as follows:

Eight days previous to admission she had a heavy, deep, dull pain in the left leg and began gradually losing control of both legs, so that five days previous to entrance—in other words, in the first three days of the illness—she had lost control of the legs to such a degree as to be unable to walk. At this time she also lost control of the bladder and bowels. The appetite is described as poor for two days before entrance. She was nauseated and vomited the day before entrance. She complained of pain in the abdominal region upon the day of entrance, about eleven hours before her admission. It was described as a sharp pain which came and went. Such was the history of the attack up to the time of entrance.

In her *previous history* it is to be noted that she had measles, whooping-cough, pneumonia, scarlet fever, mumps, and chicken-pox during childhood. She was operated upon for enlarged glands of the neck at seven years of age. In other words, it may be said

that she was unusually susceptible to the acute infectious diseases. At five years of age she fell on her head; after about three weeks she was unable to bend her back and unable to walk. She was put in a cast for six months, at the end of which time she was pronounced cured and sent home.

This history, taken in conjunction with the history of the enlarged glands in the neck, makes it very probable that the child harbored a tuberculous infection at an early age.

At eleven years of age she had three convulsions in one day, each convulsion beginning with a twitching of the left hand. She was unconscious for a time after these convulsions. There seemed to be no good reason for the convulsions so far as anyone around her can remember. In December, 1915, she again had three convulsions in one day, following which she had a severe pain in the left side of the head.

In March, 1916, she had pain in the left eye and lost the power of vision in this eye for a time. It returned, and then she seemed to lose the power of vision in the right eye. The vision has returned, but has never been as good as before the attacks.

Family History.—The parents are living and well; two brothers and one sister are living and well; one brother died at the age of three months from "gastritis."

When I first saw the patient, shortly after her entrance into the hospital, she complained of severe pain in the abdomen, in the legs, and to a lesser degree in the arms. Although no muscle group seemed to be completely paralyzed, still there was a very marked interference with the function of the legs, less of the arms. There was a slight rigidity of the neck and a bare suggestion of the Kernig sign. The mother said that previous to her entrance into the hospital there was extreme sensitiveness of the legs. At the time of my first examination, sensation, light and heavy, sharp and dull, was decreased over both legs practically up to the hips and more marked on the outer than the inner surface of the legs. Pain and touch sensations were diminished over the abdomen practically up to the line of the nipples. The abdominal reflexes were abolished, the patellar increased; there was bilateral ankle-clonus and the Babinski sign was present on both sides. The

reflexes in the arms were present, perhaps slightly increased. Pupillary reflexes and movements innervated by the cranial nerves were normal. The optic nerves, however, showed a bilateral swelling and they looked rather whiter than normal. Vision was distinctly diminished in both eyes.

During her first two weeks in the hospital the temperature remained at about 100° F., with a daily fluctuation of about 1½ degrees above and below 100° F. Eight days after her admission the temperature began to rise, and in three days gradually worked up to 103.6° F., falling within the next day or two back to the original 100° F. level, and gradually subsiding in the course of two weeks to approximately normal. During these first two weeks she complained of severe pains in both legs and feet as well as in the abdomen. At first the abdomen was rigid and distended, and for the first ten days she had to be catheterized daily. She then developed involuntaries, which continued, gradually becoming less frequent, for about three weeks. After about two weeks in the hospital she began to improve. First the pains became less severe, then movement became more and more possible and under better control, so that after about three weeks she was able to walk.

A lumbar puncture was done a few days after entrance and gave a negative Wassermann reaction, sixteen cells to the cubic millimeter, and a questionable globulin test. The white cell count on the day of entrance was 18,700. The urine was negative except that a few pus-cells were found in a catheterized specimen. A leukocyte count, made eleven days after entrance, showed 11,400. Two months after her entrance a lumbar puncture revealed the presence of nine cells to the cubic millimeter and a negative Wassermann reaction. An examination at that time showed the condition which is now present.

You see there is a suggestion of the Babinski sign in the left foot; it, however, is not at all well marked. The reflexes on the left side are a little more brisk than those on the right. Voluntary control of all four extremities seems very good. She hops and stands with closed eyes with practically normal facility on either foot. In attempting the finger-to-nose test there is a slight sug-

gestion of an intention tremor in the left arm. In pouring water from one test-tube to another she always spills some of the water, especially when the left hand is doing the pouring. The abdominal reflexes, especially on the left side, are diminished. Vision is obviously impaired, approximately only $\frac{5}{10}$ remaining. The optic nerves are distinctly blanched; their outlines are slightly blurred. They represent a secondary atrophy following a neuritis. Sensation in the legs is practically normal. I should have said that there is no disturbance of speech and none of the ocular movements. There is no mental disturbance.

Diagnosis.—When we consider the course of this disease we are forced to class it among the acute infections of the central nervous system. The rapid onset, the pains that came and went, the marked disturbance of function, both motor and sensory, that came on rapidly and disappeared almost as rapidly, the obvious swelling of the optic nerves, the febrile course, the transient interference with sphincter functions, the sensitiveness of the legs which had been present but disappeared before entrance, the nausea and vomiting, all these things speak for an acute inflammatory condition of the central nervous system rather widespread.

What the nature of the inflammatory agent is is more difficult to determine. As I said when speaking of her previous history, she showed a susceptibility to the acute infections of childhood. Also there were evidences of a tubercular infection in the body—at least I suspect this to be the case. When we consider the fact that she had convulsions beginning two years before this acute illness, then the acute illness, and then the present condition, the optic atrophy and the suggestive intention tremor on the left arm, the recurrent attacks of multiple sclerosis are forced into our minds. That is, if we consider this acute illness all by itself we are obliged to think of it as an acute myelitis, but when we consider it in connection with her previous and subsequent history we are obliged to think of it not as an incident of primary, individual importance, but as an event belonging to a series. The characteristic thing in the course of multiple sclerosis is the tendency it shows to acute exacerbations and slow remissions, the gradual progress of the disease, however, being downward. Hence, in

that respect at least, the illness of this child belongs in the category of multiple sclerosis. In multiple sclerosis some authors claim that as high as 60 per cent. of the cases show changes in the optic nerve. Usually, to be sure, these changes are in the nature of a primary atrophy, but occasionally optic neuritis and secondary atrophy are seen also. So the optic nerve changes in this little girl are such as are seen in multiple sclerosis. The intention tremor in the left arm, though not well marked, is also suggestive of multiple sclerosis.

It is simple enough to say "multiple sclerosis," but it is quite another matter to explain the underlying process which precedes the sclerotic stage. That in many cases there is an acute inflammatory period I am quite sure. It is claimed, for instance, that in every case of multiple sclerosis when there is an exacerbation of symptoms a leukocytosis appears in the spinal fluid. This is doubtless an evidence of the inflammatory condition existing within the dural sac. The micro-organism that causes such an inflammatory reaction is unknown. It is possible that it is a streptococcus, such as produces chorea, for instance.

There is one disease that we must be certain not to overlook in such a patient as this. That is syphilis. Syphilis can produce exactly this symptomatology. The optic nerves in this case especially are suggestive of syphilis. However, the age of the child, the absence of any syphilitic symptoms in other members of the family, and the absence of any physical stigmata of inherited syphilis, bear out the two negative Wassermann tests on the spinal fluid. There has also been a negative Wassermann on the blood which was done within the last few days.

Multiple sclerosis and disseminated myelitis are differentiated from each other with great care and perhaps on a questionable basis, inasmuch as neither term is etiologic, but depends upon rather slender grounds for its individuality. One might say that there is but little sense in attempting to say which of the two conditions is present in this little girl. That it is a disseminated inflammatory disease of the central nervous system involving chiefly the spinal cord and optic nerves is true without a doubt. If it were not for the history of the previous attacks of the central

nervous system—namely, the convulsions, and for the presence of the atrophic condition in the optic nerves seen within two weeks of the onset of the present attack—we might stop where we are and say that this is a disseminated myelitis, saying further that the prognosis is relatively good. But the convulsions and the eye condition lead me to believe that further attacks are to be expected such as occur in multiple sclerosis.

Consequently, a rather gloomy prognosis is to be arrived at. The tonsils have been removed with the hope that removal of that focus of infection would spare the child from further attacks. The left submaxillary gland was enlarged at the time of her entrance into the hospital; the glands in the neck are also slightly enlarged. Infection may be coming from the throat or mouth in spite of the tonsillectomy. The inflammatory reaction may be the result of the tuberculosis that we have a right to suspect is present.

Treatment.—Fresh air, good food, and avoidance of all fatigue are indicated. Hexamethylenamin in doses of from 3 to 7 grains three times daily may be of service, since there is good reason to believe that being excreted into the spinal fluid it increases the germicidal powers thereof.

Case II.—The second case, a school-girl fourteen years old, entered the hospital on June 13th. She was brought in in a comatose condition and no history was gleaned from the family at that time. Since her recovery she has told me that previous to her sickness she suffered for some time with increasingly severe headaches. She tells me that her parents are both well, that her oldest sister, now nineteen, has headaches. There are four younger brothers and one younger sister.

At the time of her entrance, as I said, she was in a comatose condition. However, it was not absolute coma; she reacted to my efforts at examination by crying out in a fretful, pitiful manner. There were some excoriations over the sacrum and sordes on the teeth. At that time there was no strabismus; the pupils were widely dilated, but reacted to light. There was rigidity of the neck. There was hyperesthesia of the body and general spasticity of the extremities; the knees were held flexed. The ab-

dominal reflexes were abolished and the deep reflexes were all brisk. There was a well-marked Babinski sign on both sides and a bilateral Kernig. The patient was making almost constant chewing motions, grinding her teeth. There was an optic neuritis, more marked on the right side.

Lumbar puncture revealed a rather thick fluid, almost of a mucoid thickness. This fluid showed about 800 cells to the cubic millimeter, giving it a slightly yellowish tinge. The globulin reactions were positive and the Wassermann was strongly positive.

At the time of entrance the temperature was normal, though it showed over a degree of fluctuation. In the first four days in the hospital the temperature gradually went up to 102° F., falling, however, in the next five days to between 99° and 100° F. rectal temperature. Here it stayed, gradually sinking a little lower during the next ten days, and finally becoming normal.

Upon entrance tuberculous meningitis was suspected. Tubercle bacilli were looked for in the spinal fluid, but none found. The *x*-ray revealed no suspicious signs in the lungs. Lumbar punctures were done every three or four days for the first two weeks. A week after entrance she was much brighter; the neck was still rigid, but the Kernig was less marked. The right plantar reflex, although very feeble, was otherwise normal.

Since about June 30th there has been steady, very satisfactory improvement, so that now she shows this condition: She is quite bright, rather vivacious, seems to be well nourished, although she is rather slender. Her gait is fairly good, but occasionally there is a slight clumsiness in walking, especially with the left foot. She can hop fairly well on the right foot, but not on the left. The Romberg is fairly well marked. There are no paralyses or pareses. The left plantar reflex shows a distinct Babinski, the right plantar is normal. There is left ankle-clonus. The right Achilles reflex is slightly diminished; the left knee-jerk is increased, the right is normal. The wrist-jerk on the left side is normal or slightly diminished, the right is distinctly diminished. The biceps and triceps reflexes are barely, if at all, demonstrable. The pupils react to light and convergence, but there is a slight

condition of hippus. Sensation seems to be normal. The fundi are apparently normal; that is, the previously existing optic neuritis has subsided.

A Wassermann reaction, done on the spinal fluid on the 5th of August, was 4 plus positive (++++). It was done with beef heart, luetic liver, guinea-pig heart, Noguchi, and cholesterin antigens. The blood has twice been negative in a Wassermann test. A thorough search for cells at the time of this second lumbar puncture failed to reveal more than one or two to the cubic millimeter.

Of course the spinal fluid findings in this case put it in an entirely different category from the first patient. The very marked lymphocytosis and the positive Wassermann speak first for a meningitis associated with the myelitis and the presence of syphilis. The most frequent manifestation of syphilis in the central nervous system is a meningomyelitis, what we may consider as a surface infection of the spinal cord or brain, with an extension into the underlying structures, the original infection occurring in the membrane covering the nerve tissue and the pia arachnoid.

In this second case also the coma is a condition not present in the first. We can explain it on two different bases: Either it is due to the infection being more generalized in this case, or to the fact that in this second case the infection is more of the surface structures, and hence interferes more with the proper absorption and elimination of the cerebrospinal fluid. In this second case the cerebrospinal fluid was under much greater pressure than in the first, so the coma may have been due or largely due to a purely mechanical cause; namely, the pressure exerted by the increased amount of cerebrospinal fluid present in the cerebrospinal cavities. We have no better history in the second case than in the first to lead us to suspect syphilis. There is nothing in the family or previous history of the child. The hymen is intact, there is no inguinal adenopathy, nothing to suggest an acquired syphilis of the usual origin. The cell count and the strongly positive Wassermann reactions, however, make it impossible for us to refuse to accept the diagnosis of syphilis. This is further borne

out by the fact that we have this peculiar combination of symptoms at the present time—the diminished reflexes in the arms and right leg, with the increased reflexes and the Babinski sign in the left leg. Such a combination is most readily explainable on the basis of a meningomyelitis in the cervical and upper dorsal cord.

Against tuberculous meningitis, which was suspected at first, is the recovery which was altogether too rapid for tuberculous meningitis—even granting that this occurs. Tuberculous meningitis and a meningitis due to pus organisms—streptococcus, staphylococcus, grip, pneumococcus, gonorrhea, meningococcus—are the only diseases we need consider in the diagnosis. We have no adequate history of the onset of the trouble such as would help out in the case of the pus organisms; that is, symptoms of abscess or constitutional infection. Her appearance upon entrance with the coma, retracted head, and hypersensitiveness left no doubt that the meninges were irritated. The lumbar puncture, revealing the thick, slightly opalescent fluid, settled the question relative to the infection of the meninges. The Wassermann reaction, of course, strongly suggested the cause of this infection. The present increase of reflexes in the left leg, the Babinski signs, and the swelling of the optic nerves all speak strongly for an involvement of the central nervous tissue underlying the meninges. The increased reflexes and the Babinski show interference with function of the pyramidal tract. The swelling of the optic nerves suggests an inflammatory condition, but may possibly be due to the increased intracranial pressure. As I said, a meningomyelitis is the commonest form that syphilis takes in the central nervous system. Consequently, we are led by the symptomatology as well as the laboratory findings to a diagnosis of syphilis, even in spite of the good family history and the absence of primary signs in the patient herself.

Treatment.—In this case the treatment must be pushed as consistently as possible. She is at present having a dram of mercury rubbed into the skin daily. The plan is to keep her on this treatment for at least six weeks, preferably two months, giving her a period of rest of two or three weeks, and starting her in on another course of rubs for a similar length of time. At the end

of this time, in case no contraindication has arisen in the meantime, she should have some intravenous injections of arsenic.

The **prognosis** is none too good for complete recovery. The disturbance of the function in the left leg is not likely to clear up entirely. It seems improbable also that such a wide-spread infection as has been present in this girl can be entirely eradicated even with the most thorough treatment. Consequently, subsequent attacks of nervous syphilis are to be expected.

CLINIC OF DR. M. MILTON PORTIS

COOK COUNTY HOSPITAL

CARCINOMA OF THE RECTUM

(OUTLINE ON BOARD.)

Carcinoma of Rectum:

(1) Varieties.
(2) Occurrence.
(3) Location.
(4) Etiology: Age, sex, predisposing causes.
(5) Symptoms:
  (*a*) Subjective: Early; late.
  (*b*) Objective: Digital examination.
    Sigmoidoscope.
    Secondary involvement: Bladder, genitalia, liver.
(6) Diagnosis: *x*-Ray.
    Tissue.
(7) Differential diagnosis: Syphilis, non-malignant strictures, benign growths, tumors outside bowel.
(8) Treatment:
  Palliative: { Medical. / Colostomy.
  Radical: Operative removal.

CASE I.—This woman, forty years of age, a laundress by occupation, an Austrian, comes into the hospital with the following history as elicited through an interpreter:

She has been sick for the past two months only and before this she enjoyed good health. Her trouble began rather gradually, with pain low down in the abdomen. She is constipated and has rectal tenesmus. She has passed no blood from her rectum. She has severe attacks of vomiting which are of frequent occurrence. The vomitus does not contain blood. There has been a slight loss of weight.

No difficulty or pain on urination have been noticed. She has no other pains; no headache; no cough. Appetite is good; bowels regular.

Past History.—Negative.

Obstetric.—One child, seven years old, is living and well. Miscarriage is denied.

Menstrual History.—Menstruation began when she was nineteen years of age. She insists that it was regular, lasting two or three days.

Family History.—Mother died of heart trouble; father died of consumption. Nothing is known of one sister and two brothers who are in Europe.

Habits.—She drinks some beer.

Physical Examination.—*Scalp.*—Negative.

Ears and Nose.—Negative.

Eyes.—Negative.

Mouth.—Teeth bad, tonsils large.

Neck.—Palpable cervical glands.

Chest.—Asymmetric; marked scoliosis in dorsal region, resonant throughout.

Heart.—Not enlarged. Tones clear; no murmurs.

Abdomen.—Soft, not tender. Liver not enlarged. No palpable spleen.

Extremities.—Tumor 2 inches below crest of tibia, 1 inch in diameter, reddish pink, not tender, freely movable. Present for thirty-seven years.

Reflexes.—Triceps, wrist, abdominal, knee-jerks present, equal, not accentuated.

Internal.—Vaginal discharge; urethra red; slight rectocele; slight laceration of cervix; uterus is anterior and freely movable. Slight mass in posterior fornix.

Rectal examination reveals ulcerating mass about the size of a baseball on the anterior rectal wall, ragged, verrucose or cauliflower-like, with a crater-like opening in it. A smaller mass is found below. It is slightly tender. A thin, reddish-pink, gelatinous discharge flows out.

Wassermann test is negative.

Blood shows a slight secondary anemia.

Urine is negative.

Patient's general condition is fair. No evidence of metastasis is present. The liver is not palpable. There is no apparent enlargement of the mesenteric glands. Slight tenderness is elicited on deep palpation over McBurney's point. The mass in the rectum is so large that it is perhaps inoperable. However,

Fig. 118.—Defect in the shadow indicated by *a* shows location of carcinoma of rectum.

the patient is very miserable in her present condition and wishes an operation.

At *operation* (September 29, 1916) a large, firm mass about the size of a goose-egg was present in the anterior wall of the rectum. It was non-movable and seemed to infiltrate the surrounding tissues. Several small hard palpable glands were present in the pelvis. The sigmoid was slightly dilated and contained consider-

able soft feces. The liver was apparently normal. No visible metastases were present. Inasmuch as radical removal of the tumor could not be carried out, a colostomy was performed.

Case II.—This patient, age sixty-five, by occupation a molder, an Irishman, comes to the hospital with the following history:

Present Complaint.—Diarrhea, tenesmus, pain in the abdomen, loss of weight.

The diarrhea began about the first of July, with six bowel movements daily, not accompanied by pain. His appetite was poor, but he had no gastric disorder. The diarrhea has continued until it reached about eight movements daily. The stools are watery. He has passed some red blood in the stool, but never very much. There is some tenesmus. He has some griping pain in the lower abdomen when he goes to stool. He appears very pale, which he dates back to the onset of the trouble. He has had daily headaches, but never severe. Loss of weight has amounted to 33 pounds.

Past History.—He was never sick in his life with the exception of an occasional cold. He had lead-poisoning five years ago when working in a brass foundry. Otherwise he has always been a strong man.

Habits.—The patient has not used alcohol to excess. He does not smoke, uses no tobacco or drugs, and is regular in his habits.

Venereal History.—Venereal disease is denied.

Family History.—His wife is living and well and has had one miscarriage. One child died in infancy. Three children are living and well. His father and mother died of old age. Five brothers and sisters are living and well. There is no tuberculosis in the family.

Physical Examination.—Patient is an adult, male, about sixty-five years old, who is very pale, but not markedly emaciated, suffering with diarrhea.

Scalp.—Negative.

Ears and Nose.—Negative.

Eyes.—Conjunctiva and sclera pearly white; pupils white and regular.

Mouth and Throat.—Lips pale; teeth bad, pyorrhea. Tongue coated.

Neck.—Large; some adenopathy.

Heart and Vessels.—Some sclerosis. Radial pulse moderately rapid, regular, and good quality. No abnormal peripheral pulsation. Heart tones distant, but present. No murmurs.

Respiratory System.—Respiration normal; chest normal; no asymmetry. Chest resonant throughout. Breath sounds rather low. Few moist and whistling râles scattered throughout lungs.

Abdomen.—Flat, muscular wall, symmetric, tympanitic in the flanks. Tenderness marked in both lower quadrants. No tumor masses; liver and spleen not palpable.

Genitalia.—Negative.

Extremities.—Negative.

Reflexes.—Normal.

Rectal Examination.—Ampulla empty; no masses; prostate large, but quite normal in consistency.

Sigmoidoscopic Examination.—Sigmoidoscope can be passed only 6 or 7 inches, and there meets with some obstruction. A tumor mass can be seen. There are no ulcerations visible. The tip of the instrument is stained with a black purulent material and there is some macroscopic blood. Rectal examination with finger is negative.

x-Ray.—Plates confirm clinical diagnosis of obstruction in the bowel. The defect in the shadow indicates a carcinoma of the pelvic colon (Fig. 119).

Blood Examination.—Red blood-cells 3,000,000; white blood-cells 18,500; hemoglobin 55 per cent.

Wassermann Test.—Negative.

Urine.—Negative.

Pathology Found at Operation.—Large carcinomatous mass at junction of rectum and sigmoid. Was not removed. Few nodules in peritoneum and metastatic masses found on surface of the liver. Some free fluid in the peritoneal cavity. Colostomy was performed.

General Considerations.—Here on the board I have given you an outline so that we can go over this subject and discuss it in a more complete way. I consider the subject of carcinoma of the rectum one of the important subjects for your consideration because, first, it is treated rather superficially in your text-books; and, second, doctors in general fail to recognize it in time. Both of these cases I have shown you came to this hospital too late for a radical operation. This is the usual sad story. The

Fig. 110.—Defect in the shadow shown by shaded line shows location of carcinoma of the rectum. Because the original plate was hazy the lower colon is marked out.

time of complaint, as a rule, dates back over months, but the cases have not been recognized in time, and the majority that come to the hospital are late cases, in the sense that they are not cases for radical operation.

Varieties.—The common variety is the adenocarcinoma. The great majority are of that type. We also get the scirrhous, the squamous cell variety, which is like the epithelioma, and, lastly, the atypic carcinoma, the carcinoma with colloid degeneration.

I will consider the last three varieties first because they are rare, and dismiss them. The scirrhous is fortunately an uncommon tumor found in the lower colon. It occurs more commonly in the sigmoid and is usually of the infiltrating type, surrounding the entire bowel wall and causing a stricture.

The squamous variety is the type seen in tumors of the rectum that originate at the anal opening. These cases resemble epithelioma. They, too, are not common.

The colloid carcinoma deserves no special comment; it occurs in all parts of the body. It is just a degenerative form.

The Occurrence of Carcinoma in the Rectum.—The occurrence of carcinoma in all parts of the body, taken together in big groups of statistics, would show about 50 per cent. in the gastro-intestinal tract. Remember that one-half of the carcinomata that you see are in the gastro-intestinal tract, and about one-third of those will be in the rectum. Therefore about 30 per cent. of the whole number of cases of carcinoma of the gastro-intestinal tract are in the rectum, and that is why we consider the subject to be so important. It is much more common than is ordinarily believed.

Location.—The majority of these tumors occur in the lower 5 inches of the rectum. You find them 2 or 3 inches above the anus, and they are very accessible to direct digital examination. They are very commonly located on the anterior wall, but may be on any part of the wall. The next common place is at the junction of the rectum and sigmoid, and that is at the height of 7 or 8 inches. This man belongs to that group. The woman belongs to the first group. Let me emphasize again that the majority of carcinomata of the rectum will be accessible to the palpating finger.

Etiology.—Usually the cases occur after forty years of age, as in carcinoma elsewhere. They do at times occur quite early, and authentic cases are found in the literature of individuals as young as fifteen or sixteen. Some cases are described in children. I ran across a case of carcinoma of the rectum described in a child of five. The majority occur after forty. The statistics show a slight preponderance in the male. As a predisposing cause we

must mention chronic inflammatory or ulcerative disease of the rectum, including hemorrhoids, fistula, and fissures. We cannot say anything of the exciting cause, for that is still a problem for the future.

Symptoms.—These divide themselves into the *subjective* and *objective*. The objective symptoms are really the things that you find on physical examination. The subjective symptoms are subdivided into the symptoms that you meet with in the early cases and in the late cases. The early cases have very few subjective symptoms at times. As a matter of fact, they are so slight that they frequently do not come to the attention of the physician in time. The patient may go about for several months with rather indefinite and uncertain complaints, and at the time he comes to the physician an examination may disclose an inoperable tumor. The symptoms may be so slight that the individual may not think it worth while to go to a doctor. What are the early symptoms? It is most commonly a little flatulence or indefinite bowel disturbance that the patient cannot always describe. He may have been in perfect health, and then suddenly begin to have an uncomfortable feeling in the bowel with flatulence and tenesmus. Laxatives and diets are tried out, and yet the symptoms persist. Commonly he will tell you that he has a pressing sensation in the pelvis. That should draw your attention at once to something serious. At other times he will come to you and say, "Doctor, I am suffering from bleeding piles." He may have had hemorrhoids for years. You look and see that he has hemorrhoids, and you are quite content. A great many of the individuals who are suffering from hemorrhoids are predisposed to carcinoma. Put your finger in that rectum and see if you can find a mass. The reason that the hemorrhoids are suddenly beginning to cause trouble is an indication that something is happening. If the hemorrhoids are of recent occurrence in an individual of forty or more, be still more suspicious of trouble higher up.

The patient comes to you, even in the early stages, with a history of irregularity in the bowel action that he did not have before. On the one hand, he will tell you he is constipated and that he has to strain a good deal at stool. The feces come out

in small masses and he does not feel that he has emptied the bowel. He may try to empty his bowel three or four times a day and still have the sensation of more to come and of something pressing. At other times he will complain first of diarrhea, just as this last patient began with diarrhea. It was looked upon as just a case of diarrhea, and the doctor made a diagnosis of "dysentery" and was content with that. That is not a diagnosis. With the diarrhea there is mucus which frequently is blood tinged. Pus may discharge, which often has an offensive odor. At times we get a combination of constipation and diarrhea. Keep in mind that alternating attacks of constipation and diarrhea occur in carcinoma of the rectum.

Later on the patients all get pain. The pain in the early cases may be very slight or absent, particularly if the tumor is located higher up. As the tumor grows and becomes fixed to the surrounding tissues, the pain increases and may become constant and severe. There is frequently very much tenesmus. The lower the tumor is located, the more pain. If it is in the anal opening they have terrific pain, as you would expect from mechanical reasons. Bleeding occurs sooner or later, and the lower the tumor the earlier does it bleed on account of traumatism by the feces. The hemorrhage is usually not large in amount, but it depends upon the type of the growth. If it is an actively growing tumor the hemorrhage may be serious. I have seen strong individuals with carcinoma of the rectum who were pulled down within a short time to a pale, anemic, and miserable condition.

The other subjective symptoms that come on late are the usual symptoms of carcinoma of any part of the body. Cachexia, loss of weight, loss of strength are commonly complained of. But these, as a rule, are late manifestations of the tumor. Toxic manifestations may be complained of due to absorption of abnormal intestinal contents. This is usually called "biliousness" by the layman, and it is entirely due to the absorption of toxic material from the diseased colon itself.

And then, among other late subjective manifestations, come the reflex disturbances of nausea and vomiting due to the ob-

struction below, the same as in chronic constipation. The woman presented this morning had nausea and vomiting until she had a colostomy performed. Among other late manifestations is intestinal obstruction, acute and chronic. The acute intestinal obstruction, of course, is serious and demands urgent surgical measures. The patient may come complaining of edema of one or both legs. If it is associated with carcinoma of the rectum it is very serious, for it speaks for involvement of the iliac vein.

Finally, the subjective symptoms may in a small number of the cases have a rather stormy onset, which is attributed by the patient to an indiscretion in diet. This may be the first manifestation of carcinoma of the rectum. So don't rule out a case because it has a stormy onset instead of an insidious one.

Objective Findings.—In the objective symptoms and signs the main thing is the *digital examination.* I would like to underscore that two or three times for you. By a digital examination in a majority of the cases you can determine quickly what the condition is, just as you did a moment ago when you examined this patient. If the patient is not too sick, the best way to make a digital examination is to have the individual stand up and bend over a table or chair, for in that way he can press down and bring the parts nearer to the finger. If that cannot be carried out, then have the patient lie on the side or assume the lithotomy position. If the tumor is not readily palpated, press in the abdominal wall with the other hand, just as you do in a bimanual examination. If there is still any doubt, make a vaginal examination besides. In this examination note the location of the tumor as well as its mobility. Also try and palpate for lymphatic involvement. If you do find something wrong by examining with your finger, or even if you don't, and the story of the patient is at all suspicious, by all means make a proctoscopic or sigmoidoscopic examination. There are clever instruments on the market which are easily handled. With these instruments you will visualize the tumor; you will see what you have, and through this same instrument it is a very easy matter to clip off a little piece. This can be submitted to a microscopic examination, and then you

have the story complete. You will know what you have; you will know the type of the tumor. Without this your examination is not finished. You will also want to know how extensive the tumor is. Has it extended to the surrounding tissue? Is it perirectal? Are the genitalia involved? And, most important of all, does the liver show metastases, for that is the common place of involvement. Palpate the liver, and if you find it enlarged, nodular, and tender, you know that metastases have occurred.

The **diagnosis** depends mainly on the objective findings. If you are in doubt about it, the *x*-ray will help you out, and will also determine the extent of the tumor. If there is a defect in the shadow, as you observed in the plate of the second patient, it speaks for a tumor. The size and shape of the growth and the width of the channel can be seen. Sometimes you can determine whether or not there is a fistula. The final examination depends on the microscopic examination of the tissue. In the diagnosis you are always anxious to know whether or not there is a secondary involvement. You must decide that to determine whether or not the case is one for radical operation.

In the **differential diagnosis** of this disease the main thing to consider is syphilis. I want to emphasize that because cases of syphilis have been overlooked. Syphilis of the rectum occurs more commonly in women, so when the disturbance is in a woman be all the more alert for syphilis. You can make a differential diagnosis by the history and by the physical findings which point toward syphilitic infection. Always examine the glandular system, look for scars and skin eruptions of various kinds; test for nervous system lesions. Finally, you have, on the one hand, the Wassermann test, and, on the other hand, the tissue examination for the crucial differential diagnosis. You should never make a mistake between syphilis and cancer of the rectum.

Non-malignant strictures of the rectum will have to be differentiated. In non-malignant strictures you will get a history of trauma of some kind or a history of some infective process. A severe colitis or dysentery may result in a stricture which you will determine from the history and other findings, or it may be

of a gonorrheal origin, and this again is more common in women. The reason for that is the frequency of contamination by discharges from the genitalia. Gonorrheal strictures are frequently mistaken for carcinoma, but the stricture of the gonorrheal type is a smooth stricture; the bowel below the stricture is usually funnel shaped and leads up to the narrow place. The mucous membrane is pale, does not bleed easily if at all, and the entire picture is that of a scar so different from the tumor that you palpate as a vascular mass, usually protruding into the bowel. The digital examination and the sigmoidoscopic appearance will give you an impression of a cervix in a vagina. But the carcinomatous mass is irregular, firm and hard, and bleeds easily. The growths that may be troublesome in differentiation are the small button-like carcinomata that you discover early. They resemble papillomata and fibroid tumors of the rectum, but they have hard indurated borders, and show evidence of involvement at the margin which is characteristic of malignant tumors. If, in addition to finding such small masses, you find evidence of perirectal involvement, and you can palpate any lymphatic glands, then you feel all the more certain that the little mass is not a papilloma or fibroma. You must, however, not stop there. Take out a little piece and put it under the microscope.

Treatment.—We have to consider the palliative treatment and the radical or operative treatment. When a case comes to you for treatment it frequently will come with the story of bleeding piles or fissures. Make a complete examination, for I am sorry to say that surgeons have operated for these conditions and have not even examined the bowel when a carcinoma has been there all the time. So I want to emphasize in the treatment of your cases that you make in every instance a thorough examination, and when you determine that a carcinoma of the rectum is present the next thing you must decide is whether or not it is operable or inoperable. There are various ways of attacking these tumors, either by the perineal route or resection of the sacrum, or by laparotomy. Whatever operation is to be carried out is a matter for the judgment of the surgeon. Many surgeons perform a preliminary colostomy, so that plastic work to join the

upper and lower segment will not be necessary. In other cases is is perfectly feasible to use the anal opening, and that is by far the route to be preferred.

In the cases that cannot be operated on what are we going to do?

I believe that it is wise in cases with a purulent bloody discharge to do a colostomy, because you save the patient a lot of pain and a lot of annoyance with these foul discharges. In cases with obstruction a colostomy must be done; there is no alternative. A patient may then go along for several years with comparative comfort. Colostomy gives relief from the local trouble. Of course, it involves the trouble of caring for an artificial anus. It is surprising how soon the patient learns to care for this himself, and he has less discomfort than you would think.

Otherwise the medical treatment is purely palliative. You choose a diet that does not leave much mass for the stool. Give a diet of milk, eggs and fruit, some meat, but avoid the coarser vegetables and fruits. Give the fruit juices, orange and grapefruit. Custards and strained cereals may also be used. If a colostomy has been done the patients may eat what they please.

The rest of the treatment is purely symptomatic. Give something to control the pain and give the patients as much as they need to keep them comfortable. The opiates deserve first place. The salicylates will give relief, and at times belladonna is of service. Such patients must, of course, have tonics, principally iron and some arsenic, if they become anemic and cachectic. But above all things, when you have an inoperable carcinoma of the rectum, and you know it is carcinoma, try to make the patient as comfortable as possible and do everything and anything you can toward that end.

The feasibility of deep Röntgen therapy and radium therapy must also be considered in the inoperable cases.

CLINIC OF DR. SOLOMON STROUSE

MICHAEL REESE HOSPITAL

THE DIAGNOSIS OF EARLY ACTIVE PULMONARY TUBERCULOSIS

Symptoms and Signs of Value in the Diagnosis of Incipient Pulmonary Tuberculosis. Careful Routine History and Examination Essential in All Cases. The Role of Tuberculin and the Röntgen Ray. Prolonged Observation Often Necessary Before Arriving at a Definite Diagnosis. Clinical Picture Simulating Hyperthyroidism.

IN the clinic today we shall devote the whole hour to a consideration of the diagnosis of early active pulmonary tuberculosis. Instead of presenting numerous cases with complete case records, I shall simply use the patients whom you see before you to illustrate various points as they are brought up in discussion. It seems to me that one is justified in stopping long enough to consider this question in all its phases, and to analyze and weigh the many factors that are of importance in establishing a diagnosis. Of course, when a case of tuberculosis has advanced far enough to offer definite pulmonary signs, or to show tubercle bacilli in the sputum, a diagnosis can be made with no difficulty, but the fact that there are so many signs and symptoms, so-called, of incipient tuberculosis is proof enough that there is no specific means of diagnosing tuberculosis early in the disease. In fact, a rather extensive experience in handling such cases forces me almost to believe that the diagnosis of incipient tuberculosis is beyond the present means at our command. This may sound like heresy, but I think when we present these cases and bring out the various points you will agree with me that there is some justification for this statement.

Take this first patient, for instance, a young man twenty-five years old, who comes for an examination because he has not been "feeling quite well." His history is that for the past month or so he has noticed an absence of his usual vigor; he has a tendency to flush in the evening, but he has never taken his temperature; he tires easily. Otherwise he has no complaints.

His past history is not perfectly normal; he has been a strong and healthy boy; he has worked and has been, on the whole, a great deal out-of-doors. His father died of pulmonary tuberculosis five years ago; his mother now has a moderately advanced case of tuberculosis. The boy was exposed until within a few years to the disease, but since that time has been living away from his mother, and has taken unusual precautions in the way of out-of-doors and healthy living and proper dieting and exercise.

On examination you see a strong, healthy man. At this time (11.00 A. M.) his temperature is 98.6° F. and his pulse 88. His nourishment and musculature are excellent. His mucous membranes show no pallor, his hemoglobin being, as a matter of fact, 90. His teeth and pharynx are normal.

Now let us strip him and examine his chest carefully. But before examining him let us emphasize this fact, that the only way to examine the chest is on the bare skin. An undershirt, even when made of silk, is a barrier to the passage of adventitious sounds or a factory for such sounds. The patient must also be put in the proper light. He must be facing the light, so that whenever possible it will strike him broadsided, and there will be no shadows on one side of the chest which are not present on the other. Examination of the chest is best conducted in a routine manner which is followed in all cases. Careful inspection is verified by palpation, which often will point to the seat of trouble at once. A lagging apex or base will at once focus attention on the spot; slight differences in contour of the two sides of the chest may be noticed when percussion and auscultation give only vague indications. In palpation, I find considerable help in carefully noting the muscle resistance, although I do not feel capable of drawing the conclusions from such palpation that Pottenger does. In percussing the chest both light and heavy strokes can be used to

advantage if properly interpreted; likewise in auscultating, the patient should breathe in his normal fashion first and then should take deep breaths. In bringing out râles the short sharp cough at the end of inspiration is invaluable. Perhaps the best reason for the adherence to such a routine is that it gives you a normal basis with which to compare abnormal findings. The more chests one examines this way the more does the examiner become accustomed to *normal* variations, and that, of course, makes the discovery and interpretation of the pathologic much less difficult.

Now let us apply these simple rules to our patient. We see immediately that there are no particular changes in the contour of his chest, that there is no retraction of his apices, and both sides move with equal freedom. If you place your hands on his chest and watch carefully the respiratory movements you will notice the same freedom of motion. There is no muscle resistance and no differences palpable in the depths of the supraclavicular fossæ.

On percussion, we find a very slight dulness in the right supraclavicular fossa and even as far as the second rib. This dulness is also found posteriorly in the supraspinous fossa and also slightly along the right side of the vertebral column. Otherwise the percussion note throughout is resonant.

Auscultation shows over this area a very slight change in the expiratory and inspiratory ratio. In other words, expiration is a little prolonged, but the breathing can hardly be called bronchial, much less tubular. No râles can be heard even after repeated coughing. You notice also that over this slightly dull area the whispered voice sounds are a little more intense than over the rest of the lung. The vocal fremitus is also slightly increased.

In the remainder of the examination we note that the heart, abdomen, reflexes, etc., are all perfectly normal. There is no glandular enlargement. The boy has no sputum, so I can give you no report on that. The blood and urine are normal.

I know of no better example upon which to hinge a discussion than this patient. His family history is, to say the least, most suggestive. His parents both have tuberculosis, to which he was exposed in infancy. Although we are inclined to consider inher-

ited pulmonary tuberculosis as a very rare anomaly indeed, we are far from being as optimistic about the possibility of direct contagion from parent to child. The fact that tuberculosis is common in families; that it may go from generation to generation, is proof enough that the idea of a familial tendency in the disease has some importance. This familial tendency is almost always direct contact. If a child of tuberculous parents is taken away from his parents at once and placed in healthy surroundings I think his chances of not getting tuberculosis would be essentially greater than would be the child of healthy parents placed in unhygienic surroundings or in a home in which tuberculosis was or had been present. Therefore in the case before us we must lay considerable stress on the family history as a positive element in diagnosis.

If this patient were, for instance, a tailor or stone-cutter, or had some occupation which would render his resistance lower than normal, the family history would be of even greater importance, but, as we emphasized in his history, he has lived and worked outdoors. We know that groups of workers in certain trades seem especially prone to pulmonary tuberculosis, as, for instance, workers in the tailor trades. What is there in the tailoring job that renders a man especially susceptible to this disease? Isn't it really a question of unhygienic surroundings? Isn't it a question of hard work in a poorly ventilated shop, with insufficient food, insufficient air, lack of exercise, and poor surroundings at home? If the tailor were working in a well-ventilated shop and if his home surroundings were good, I see no reason at all why he should be so especially subject to tuberculosis. On analysis, then, the predisposition of the tailor to tuberculosis is more a question of how he works, and the modern battle against tuberculosis, by getting at the root of things, insisting on better working conditions and better living conditions, will ultimately show that the tailoring trade *per se* is not essentially harmful. But in obtaining a history it is extremely important to learn just how the patient works and lives as well as how he eats and sleeps. In this patient, of course, this is a negative factor, but the mere fact that he is not in a trade which is supposed to be

predisposing to tuberculosis should not be held as evidence against a diagnosis.

As far as his past history is concerned it is perfectly normal. Questioned as to having infection of the tonsils, infection of the nose or throat, he denies any such thing. He has never had attacks of influenza (so-called), of pneumonia, or of any disease of the respiratory tract.

On the other hand, this little girl who is standing alongside of him comes in because she is coughing; states that every year she gets attacks of "grip." When you analyze the "grip" you will find that it consists of a cough and being "run down." She has never been thoroughly examined during these attacks, and yet one cannot help but feel off-hand that such frequent attacks of infection in the respiratory tract are probably tuberculous. It is very common, indeed, to meet patients like this little girl, and the sad part of it is that when finally they do get examined they have a very definite pulmonary tuberculosis.

Allow me to digress a moment on the importance of great accuracy in history taking. Patients have very poor memories, but they are likewise very susceptible to the influence of questioning. It is not at all difficult for two different men, taking the history of the same patient, to obtain two entirely different stories. This is especially true among people of foreign birth, whose knowledge of the language is not particularly good. But it can happen even with patients born and raised in America who know the English language perfectly. To my mind there is only one way of taking the history accurately, and that is to go about it without any attempt to prove anything and to stick as nearly as possible to a routine in all cases. If a patient tells you he has a cough, that is no reason for assuming that he has tuberculosis and, therefore, trying to prove it by his history. It is a reason, however, for the most extreme care in the attempt to elucidate the absolute truth.

If this is true for the family and past history, it is essentially true for the history of the illness of which the patient complains. This young man does not complain of cough, nor did he when questioned repeatedly admit having coughed. So we can assume that

he has not been subject to coughing. On the other hand, just as a diabetic forgets when he is passing urine with increased frequency, so the man who is coughing is likely to deny that he ever coughed, while at the same time he will expectorate in your presence. He accepts his cough as part of himself and does not bother about it, and in giving you a negative history he is likely to be overlooking the very point you are trying to find. He is much more likely to complain of *pain* in the shoulder or chest.

Symptoms of Incipient Tuberculosis.—We hear a lot about the *pretuberculous stage of tuberculosis*, in which the patient has various symptoms referable to different parts of his anatomy, but does not cough. We hear of the young girl who has "trouble with her stomach" and the man who is "unable to work." That such cases occur is certainly true, and I have two patients here today who have undoubted tuberculosis, and yet who have never coughed and are not coughing now; but in by far the largest percentage of cases one of the early symptoms of pulmonary tuberculosis is cough, and with the cough usually goes expectoration. This may only be a slight hacking cough in the morning with one small piece of sputum, or it may be more or less intermittent, occurring only on irritation. Then we see patients, such as this young lady to the right, who in perfect health cough once, have a hemoptysis, and are found to have typical lung findings of pulmonary tuberculosis. The blood-streaked sputum is a sign, I think, of less value than is generally accredited to it. In dispensary practice especially patients come in complaining of spitting blood, will show you blood-streaked sputum, and yet, after years of observation, will have no active signs of pulmonary tuberculosis. I have under observation now at least fifteen or twenty patients in the dispensary who repeatedly have bloody sputum, and yet in none of them have tubercle bacilli been found; in none have any signs of activity in the lungs been evident. In two or three of these patients there has even been considerable doubt as to the diagnosis. Still, blood-streaked sputum, generally speaking, should be considered a sign of tuberculosis unless proved otherwise. On the whole, cough and expectoration must be taken as the most

reliable indicators of trouble; neither cough nor expectoration is, however, pathognomonic of the disease.

How about the other symptoms?

Loss of weight, loss of strength, pains, fever, gastro-intestinal disturbances, are these signs of tuberculosis? Most of them are not. Most of them are attributable to low-grade chronic infection. A young man with pus in his tonsils, and absorption from this pus might give all the symptoms just enumerated. A woman with a pelvic inflammatory disease would be very likely to lose weight, to have evening temperature. In other words, most of the symptoms of tuberculosis that one encounters are certainly *not specific reactions* of the body to the tubercle bacillus, but are simply *general reactions* to a chronic infection. It is true that tuberculosis is probably the most common infection of a chronic nature that one encounters, so the present chain of symptoms might be considered *a priori* evidence of the presence of the most common chronic infection—tuberculosis.

So, then, we can see that as far as the history is concerned many important data can be obtained from an accurately given statement of fact. Patients who complain of one or of a group of the symptoms spoken of must be considered as possible harborers of tuberculosis, and must be examined with the view that they may have the disease, but we ought never to overlook the possibility of some other chronic infection being the causative agent.

Now we can discuss the **physical findings.** In going over this young man with you I have simply indicated a *careful routine examination.* As you know, literature on tuberculosis is crowded with signs named after various men who think their sign is individually of great importance; yet I consider it illogic to use any man's name in describing physical findings. The use of a man's name in connection with signs or symptoms has a tendency to draw one's mind away from the logical path of pathologic physiology or pathologic anatomy, and has a tendency to make one rely on points of minor importance rather than on the composite picture obtained by general careful examination. What do the signs in this patient signify? Do his history and his

physical examination combined spell tuberculosis? It is true he has dulness; he has changed breath sounds in the right apex, but what are the possibilities that might be causing those changed signs? You all know that it is practically impossible to get a right apex corresponding with the left in full degree; dulness with changed breath sounds is normally present in many persons at the right upper lung. On the other hand, a second possible cause would be an old lesion. This patient may have had tuberculosis when he was younger which has entirely healed. In the third place, the signs may be the result of a non-tuberculous infection, and in the fourth place the signs may be the very early signs of pulmonary tuberculosis, because it is true that the right apex is particularly prone to the attacks of tubercle bacilli, and the signs elicited in our patient *are* signs of early involvement.

Now the great difficulty in a case such as this is just how to interpret such signs. Even were there a few râles present the signs by themselves would simply mean some anatomic change in that part of the lung. We are not particularly interested in diagnosing tuberculosis as an anatomic condition. We want to know if this patient has tuberculosis that at present is active and causing his symptoms. If the statistics of the postmortem room are correct that over 90 per cent. of all persons have tuberculosis at some time or other, and if our statement that the right apex is particularly subject is borne in mind, the difficulties met are even greater. And yet a vast majority of the really early cases of tuberculosis will present no more marked findings than this young fellow presents.

Have we any other means of assistance in getting at the truth? Personally, I do not believe that any special physical signs mean anything more than anatomic changes in the lung, and I do not think we can emphasize too much that our interest does not lie in anatomic changes, but in a tuberculous infection. How, then, will we handle this patient? What will we do to make a diagnosis in this case? Have we exhausted all our technical means in diagnosing?

Let us take this patient to the fluoroscopic room and see what the lungs look like under the *x*-ray, and then we will have a

stereoscopic picture of the chest made which we can show you at the next clinic.

Fluoroscopic Examination.—In looking at the patient through the fluoroscope you will notice that there is a slight dulling of the right apex; and when he takes a deep breath the right apex does not clear quite as well as the left. You will notice that his heart is in normal position and is beating rapidly, probably on account of the excitement. Both diaphragms are in normal position and move freely on respiration. The details of the bronchial tree are not very clearly brought out in the fluoroscope. So far we have only proved what the physical examination showed. The slight dulness of the right upper simply means a thickening of the lung in that area, but does not mean that the thickening is due to an active tuberculosis. The *x*-ray plate which will be taken may help us considerably, but we will discuss that in a few minutes in connection with another patient.

Tuberculin in Diagnosis.—Should we give this patient tuberculin? On this question opinion differs. Personally, I think that we are far from justified in giving a diagnostic dose of tuberculin to such a patient as we have just seen. The two most important reasons for this opinion are that, in the first place, tuberculin does not definitely prove the presence of anything more than a tuberculous lesion, and does not prove that the change in the lung is active and causing the patient's symptoms. In the second place, tuberculin may light up an old lesion, and may cause more trouble than is warranted by the knowledge gained from its use. Personally, I rarely use tuberculin in diagnosis. There are occasional cases where, under hospital observation, all fine points of diagnosis are gone into and nothing definite proved, and where it is of vital importance to know whether the patient has a tuberculous focus. In such cases it might be advised, but where you can keep the patient under observation its employment as a routine practice seems inadvisable. When you do use tuberculin, which one of the many that are now on the market should be employed? I think that common practice nowadays has reverted back to Koch's old tuberculin (O. T.), because it seems doubtful whether any of the other products of the tuber-

cle bacillus have any greater diagnostic value than O. T., and from the standpoint of comparative statistics the employment of a uniform substance would seem indicated. If we had to use tuberculin, then, I should use O. T. and should start with a subcutaneous injection of 0.2 milligram. As you all know, the temperature should be taken at two-hour intervals, forty-eight hours before and after the injection, and the injection should not be made if the temperature is above 99° F. at any time before the injection. Likewise, the chest should be examined frequently, because in addition to the possibility of a general reaction one must always look for a focal reaction. If the first dose produces no reaction, we can next give 1.0 mgm.; then 2.0 mgm. Some men give as much as 5.0 mgm. Except in young children the use of tuberculin in other form excepting subcutaneously is of even less value. As is well known, with children under ten years of age the von Pirquet skin test is of the greatest value, because if children under ten years of age have tuberculosis the chances are that it is active. But the same lesion in a child of five that could give a positive von Pirquet at five would give a positive von Pirquet at thirty-five when the patient had completely overcome the original infection.

Röntgen Ray Signs.—We mentioned a moment ago the *x*-ray in connection with diagnosis of pulmonary tuberculosis. Let me show you a patient in whom the *x*-ray proves clearly the presence of an active tuberculous lesion, and let me show you the plates, so that we can discuss them. This young lady has had a cough for some time with a little fever; there had been no exposure to tuberculosis as far as was known, but the young lady was a music teacher who lived indoors, did not eat proper food, and lived under unhygienic surroundings at home. She is twenty-seven years of age, and noticed about a year ago that she was not able to work as hard as she had been previously. Very shortly thereafter a slight cough developed in the morning with expectoration. The sputum did not contain tubercle bacilli. The patient has a headache, lassitude, and rise of temperature in the evening (99.6° F.). She shows on physical examination slight retraction of the right apex, with dulness, rough breathing with prolonged expiration,

and a few râles at the end of inspiration. Of course, with this history and this examination one is justified in making a diagnosis of pulmonary tuberculosis without the presence of tubercle bacilli, and it is interesting to note that the *x*-ray plates are perfectly typical of a tuberculous lesion that is, however, no longer incipient.

Putting these two plates in the stereoscopic holder, you can see that branching out from the fundus of the right lung is a heavy meshwork containing a few small round solid areas. This branching continues practically to the periphery, where the fine lines come together to form indefinitely outlined spaces. The branching extends up to the outside border of the right upper lung, verifying our diagnosis.[1]

The value of the *x*-ray in diseases of the chest of all sorts is simply tremendous, and the more one uses it as a diagnostic means the more value is one inclined to attach to it. This does not apply only to tuberculosis, but to the differentiation of all sorts of obscure chest conditions. I have seen large, deep-chested men, in whom physical examination made by different physicians was negative, and yet who showed a very clear-cut and definite lesion under the *x*-ray. One man whom I recall particularly had an hemoptysis with an absolutely negative physical examination. Stereoscopic *x*-ray examination showed a rather advanced and extensive tuberculous lesion of the chest.

I had another man who weighed over 200 pounds, whose history sounded very suspicious of lung abscess, whose physical examination showed merely the slightest dulness on percussion of the right lower lobe. The Röntgen ray, however, showed a large abscess extending up along the mediastinum.

In pulmonary tuberculosis the Röntgen ray is of value not only as an aid in diagnosis, but often as a means of excluding tuberculosis. When I first began to compare physical findings with *x*-ray findings in the way of Wolman and Dunham, at Johns Hopkins, I was surprised by the fact that careful examination

[1] Owing to difficulty in reproducing chest plates so as to show the detail necessary for diagnosis, it has seemed best not to attempt their reproduction. Properly to interpret Röntgen readings of the lungs requires the original negatives.

of the chest usually produced about the same results as an *x*-ray, but in several instances small cavities were shown by the *x*-ray which physical examination, made even after we knew that there was a cavity there, failed to show. It is rare, indeed, for physical examinations to show more than the *x*-ray shows. It is not rare, however, for the *x*-ray to extend one's observations, but I have seen one particularly interesting case in which the *x*-ray was of the greatest value in excluding a diagnosis of tuberculosis. I brought this boy here today to show you. He is twenty-two years old; I have had him under observation for four years. When he came to the dispensary four years ago he complained of hemoptysis, loss of weight, fever, and inability to work. He had been a healthy young man up to the onset of the illness; had always been of a neurotic type and was inclined to be quarrelsome. There was no tuberculosis in his family; he was by occupation a peddler, living an out-of-door life. He was unmarried. He had, as far as we knew, no source of worry to make him nervous.

On *physical examination*, he was a rather flushed individual, with marked cyanosis of his hands and feet; his temperature was 100.5° F.; his pulse was 104. He had a marked tremor of his hands; his eyes were slightly prominent; he had a definite thyroid enlargement. Examination of his nose and throat was negative. He had no enlarged tonsils and no evidence of infection anywhere in the upper respiratory canal.

Physical examination of the chest revealed a definite, fairly large-sized area in the right upper, in which there was dulness down to the third rib, suppressed breath sounds with prolonged expiration, and some fine crackling râles at the end of inspiration. The rest of his lungs were normal.

His heart was enlarged about a centimeter to the left. The sounds were clear and very thudding and the pulmonic second was accentuated. Nothing abnormal was made out in the abdomen. The reflexes were markedly exaggerated and dermography was present.

Incidentally, you can see all these signs on the patient today if you wish to take the time to examine him. Therefore with the history and physical examination, the diagnosis of pulmonary

tuberculosis with hyperthyroidism was made. For four years the patient was treated at various sanitariums and in the dispensary. During all this time he has shown at every visit to the dispensary a temperature of 100° F. or higher, with a pulse usually between 100 and 120. There has been no loss in weight, no change in the physical condition, and no change in the signs in his lungs, except that the râles have disappeared. A few months ago when I saw him I began to doubt my diagnosis of pulmonary tuberculosis. The hyperthyroidism was undoubtedly present, but the signs of hyperthryroidism are not infrequent even in the incipient pulmonary tuberculosis. The presence of hyperthyroidism with signs of pulmonary tuberculosis has impressed itself so firmly on my mind in dispensary practice that I feel that there must be more than a casual interrelationship between the two. Of course, we must not forget that the early symptoms of pulmonary tuberculosis may include tachycardia, nervousness, loss of weight, etc., just as in hyperthyroidism, but in many cases I have seen goiter, slight eye changes, tachycardia, and tremor present with signs of an incipient tuberculosis, and I have seen these signs of hyperthyroidism clear up completely as the tuberculosis improved. Considerable literature has been written on this interrelationship. It will not pay us to go into detail today, but it might be well to remember that there are certain French clinicians who do not believe that fever ever occurs in a pure hyperthyroidism, and who do believe that the presence of fever indicates a tuberculous infection.

In this patient, then, the presence of hyperthyroidism did not in any way influence us against a diagnosis of tuberculosis, but when I saw him a few months ago, and stopped to figure that pulmonary tuberculosis of four years' standing, active enough to produce fever, tachycardia, and all the other signs this patient had should have caused a more pronounced change in his general well-being, I began to doubt the validity of the diagnosis.

The same reasoning would apply as far as diagnosis of exophthalmic goiter is concerned. Exophthalmic goiter, if severe enough to produce all the symptoms that this boy had, should have made more pronounced changes in him.

I sent the patient into the hospital for observation. Twenty-four hours after his admission I went up to see him and found he had a perfectly normal temperature and a pulse of 72. In fact, during his whole stay of one week in the hospital he never once showed either fever or tachycardia. The *x*-ray in this case was very carefully made, and showed absolutely no signs of tuberculosis. The changes in his right apex were undoubtedly due to anatomic anomalies, and were probably never an indication of a tuberculous lesion. After having him in the hospital for a week, finding everything negative, we were forced to the conclusion that this patient's symptoms and signs were the result of a nervous irritability of an unusual nature. His acrocyanosis, his general nervous instability, his disposition, all were supporting evidence of this diagnosis. He may belong to one of the as yet obscure groups of diseases produced by disturbance of the ductless glands. You will be interested to know that since we were convinced that our diagnosis was wrong, and told the patient to get out and hustle for himself and quit coming to see us, he has for the first time in four years been able to earn his own living.

But let us now return to our first patient and summarize the whole discussion. What are we going to do with him?

I shall keep this boy under clinical observation for one or two weeks. I shall watch his temperature, pulse, and weight curves very carefully. I shall perhaps give a small dose of potassium iodid and see if we can produce expectoration, and then examine the sputum most carefully for tubercle bacilli. If during this week I find that he has a temperature of 99.5° to 100° F. in the evening, if I find that he has no other source of infection, that especially his upper respiratory tract is free from any possible lesion which should cause fever and the symptoms he complains of, then I should make a diagnosis of *probable incipient pulmonary tuberculosis*. However, I should still be extremely cautious, and while I should treat the patient for pulmonary tuberculosis I would feel that perhaps it would be doing him an injustice, and before the final label I should really await the best test of all, the test of *time*.

Since we have not any absolute symptoms or signs to make a

diagnosis of incipient tuberculosis, we are compelled to fall back on the clinician's greatest asset—his common sense and his judgment. What would you do if you thought that you had tuberculosis and yet were not sure of it? Would you do what the doctors did ten years ago when the first sign of trouble was revealed, drop your life's work and get away on the assumption that you had a disease whose existence you could only suspect and not prove? You might do that if you had plenty of money and were ready for a vacation anyhow, but would you do it if your existence depended on your income? You would probably think twice. And I want especially to plead for a second thought before labeling as a positive tuberculosis the patient whom we can only suspect. We can handle him without any danger to himself and to anybody else if we keep him under clinical observation while we are making the diagnosis. Ten years ago, when percussion and auscultation had reached a stage of overacute development, many persons were diagnosed as tuberculous and sent to sanitariums for conditions which, as their future histories have shown, could hardly have been tuberculous. Several of my classmates at college lost a year when it was most needed, and subsequent history has proved pretty definitely that in all probability the trouble was never tuberculous, or, at least, if tuberculous, not of sufficient severity to warrant such a sacrifice. Today the same men would be kept under observation and would be carefully examined from time to time. Then a catarrhal apex which occurs more frequently than we suspect will clear up, and we will find that a patient did not have an active tuberculous focus. And in the use of the term "active tuberculosis" lies the chief source of our diagnostic difficulties.

To summarize the situation, incipient tuberculosis is a disease with a vague history, which may or may not definitely include the respiratory tract, and with pulmonary signs often so slight as to evade the ability of even the most acute clinician. Autopsy records show such a high incidence of healed pulmonary lesions as to mean only one thing—that most persons at some time or other have been infected. Carrying the argument still further, this infection may or may not have produced symptoms, may or may not have been progressive. If it produced symptoms and was

progressing, it was probably diagnosed during life; if it produced symptoms and then regressed, it may very well have escaped diagnosis. The pathology of an early tuberculous lesion is such as to make one doubt whether it could possibly be discovered either by the most astute clinical sense or even by the Röntgen ray. When the tubercle has grown it will give physical signs, but no longer the signs of incipient tuberculosis. There is nothing specific in the history, no pathognomonic sign of active tuberculosis except the tubercle bacillus. The variations found in many normal chests give physical signs indistinguishable from those found in a patient whose subsequent course proves these signs to indicate early tuberculous changes.

There is then no great possibility of diagnosing incipient pulmonary tuberculosis as an active condition producing symptoms. The best you can do, if you want to give full justice to your patient, is to suspect it; to watch the patient as carefully and as closely as possible under conditions which will not harm him if tuberculous, and yet will not force great social and economic losses on him. If he had an active tuberculosis you will know it soon enough to give him full benefit of proper treatment.

There is another side to this story which many men hold. Since at some time or other almost every one has pulmonary tuberculosis, every one who is "suspicious" should be diagnosed as tuberculous. While this point of view from a purely scientific basis may be as tenable as the one I have just given, it is entirely impractical from a social or economic standpoint, for if it were adhered to most of us and our friends and patients would be "taking the cure." But whichever side is adhered to it must be remembered that the earliest possible indications of pulmonary tuberculosis will be grasped only by most painstaking history, and most acute physical examinations in which process our senses are sharpened to detect the slightest changes. Then add logical deduction and sound common sense, and the result will be neither wild branding of all suspicious lungs as tuberculous nor negligence and delay in diagnosing tuberculosis when it exists.

We shall have to leave the Treatment of Pulmonary Tuberculosis for a later clinic.

CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

GASTRIC ULCER WITH INTERVALS OF LATENCY. HISTORY OF REPEATED GASTRIC HEMORRHAGES

Differential Diagnosis from Duodenal Ulcer. Treatment of Bleeding Ulcer.

November 23, 1915.

THIS patient entered Mercy Hospital on November 16th. Before reading his history I will call your attention to his pallor, and to the fact that the pallor has a lemon-yellow tinge, which suggests an extreme anemia. One cannot say by merely looking at him whether he has pernicious anemia or secondary anemia due to lead. Many lead secondary anemias give a yellow color combined with pallor which is very like the color this man shows.

Intern's History.—On October 5, 1915, the patient had a profuse bowel movement of a watery, brownish-black material; this occurred at about 3 P. M. At 12 midnight he vomited a moderate amount of material which resembled the above in that it was rather brownish, but not black. While going to the toilet he became weak, felt dizzy, everything before him became black, and he fell in a sort of faint. He says that he did not entirely lose consciousness; the bowels moved and he returned to bed; he subsequently broke out into a cold sweat and vomited again.

Such a history can indicate only one thing, and that is hemorrhage, and it is extremely characteristic of hemorrhage in both duodenal and gastric ulcer. We have seen several cases of ulcer with just such a history, the hemorrhage occurring often at midnight. (Just recently we had a case in a young man who fell in a faint on the way to the toilet-room in the middle of the night.)

In the morning our patient called the doctor, who gave him

some medicine, which stopped the vomiting until 4 o'clock in the afternoon, when he again vomited twice, the material being just about the same as that vomited at midnight. At first sight it might seem that the vomiting of brownish blood indicated an ulcer of the duodenum rather than of the stomach, because one would think that if he had a gastric ulcer and it had suddenly begun to bleed he would vomit bright red blood. It does not prove, however, that it was not a gastric hemorrhage, because blood can remain long enough in an acid stomach to become brown by the action of the acid before being expelled. The color of the vomited blood, therefore, tells us nothing as to diagnosis. He had had no pain anywhere, either preceding or during this attack.

He went to a hospital after this; he did not vomit again, but a blackish material came away from his bowels twice afterward as the result of an enema. This is again the usual history, because the hemorrhage took place when he got up in the night and fainted, and blood did not all pass out until the enemas were given; it remained in the intestines until that time.

DR. MIX (to Patient): Can you tell us when it was the bowels moved after you went to the hospital? Was it the next day?

PATIENT: It was on the 7th.

DR. MIX: So, by way of summary, he was taken ill at 3 o'clock on the afternoon of the 5th; at midnight on his way to the toilet he fell in a faint and had a rather black bowel movement, broke out in a cold sweat, and vomited; on the 6th he vomited some more in the afternoon, and on the 7th, after reaching the hospital, he had a black bowel movement as the result of an enema.

(To Patient): How black were these bowel movements?

PATIENT: They were not really black, but they were dark.

DR. MIX: Indicating, then, not so severe a hemorrhage as occurs in some cases where the bowel movements are tarry black. Were they very foul smelling?

PATIENT: No.

DR. MIX: Ordinarily they are very foul smelling. Were the stools formed, slushy, or fluid?

PATIENT: They were rather slushy, kind of thin.

DR. MIX: A very profuse hemorrhage gives a tarry-black bowel movement which is sticky and very foul smelling. We may conclude from his statement that the amount of hemorrhage into the bowel was not so great as it is in some cases. Furthermore, we recall that he just barely fainted when the hemorrhage occurred. He suffered with severe headache for several days afterward, shortness of breath, and palpitation of the heart. This makes absolutely complete the history of the hemorrhage. There might have been one other symptom, and that is, that immediately following the hemorrhage there is often a bradycardia, which later gives place to a tachycardia, the pulse becoming rather rapid. This symptom on the part of the pulse is associated with the headache, dyspnea, and anemia of hemorrhage from ulcer. During the summer preceding the patient had indigestion. Gas would form in his abdomen and his stomach would seem to tie itself in a knot; he never vomited and he never had a distinct pain or burning sensation.

(To Patient): When your stomach used to tie itself into a knot, did it hurt?

PATIENT: No, no; a knot would seem to form and then I would belch and it would be gone.

DR. MIX: Evidently he had a feeling that he must belch and that belching would relieve him, but he could hardly have had a true pyloric spasm, since pyloric spasms are very painful. He has been well since November, 1914; on that occasion he vomited a small amount of dark red blood and he passed blood in his stools. They were about the same color as on this last occasion.

(To Patient): Did you, in November, 1914, have some of this same feeling of the tying in a knot or was there pain at that time?

PATIENT: I never have had any pain at all.

DR. MIX: His third attack, going backward, was on December 10, 1909. He vomited blood in such large amounts then that he did not recover his strength until the following spring. This blood was also dark and much the same as in this last attack.

(To Patient): Did you get as pale then as you are now?

PATIENT: Yes.

DR. MIX: Did you have the same symptoms?

PATIENT: Yes; just about.

DR. MIX: Then three times this same sort of thing has happened to him. During or preceding these attacks—December 10, 1909, November, 1914, five years later, and November, 1915, one year later—he had no pain, and he never had nausea or vomiting between the attacks. In the intervals between them he has been entirely free from symptoms. His appetite is good, but his bowels move only every three or four days; he is markedly constipated. He arises with a bad taste in his mouth; his teeth are in good condition; he is not subject to sore throat.

An interesting point is that all of his attacks have occurred in the fall of the year. The contention which Rosenow makes is that ulcers are embolic in origin and due to infection. It is, therefore, not strange that they occur in the season of the year when infections are most prevalent. Hence it is that there are spring and fall exacerbations of gastric and duodenal ulcers, and this man has had all of his attacks in the fall. It seems to be a fact that in the fall of the year there are more cases of duodenal ulcers in the hospital than in other seasons of the year. He had measles at twelve years of age, chicken-pox at six. There is no venereal history; the only other significant fact in his history is that he is subject to colds in the head. He is unmarried. His father is living at fifty-two, his mother is living at fifty, and both are well; he has one brother living and he lost one at the age of eighteen from typhoid fever.

The **diagnosis** is a question of localization, whether it is a gastric ulcer or a duodenal ulcer. The duodenal ulcer is usually more positive in its work; 93 per cent. of these ulcers show pain. Of the other 7 per cent. I would say that all show distress. The pain or distress comes on at varying lengths of time after the meal, usually in one to two hours. I believe that there is a relationship between the appearance of the pain and the location of the ulcer. The nearer the ulcer to the pylorus, the quicker the appearance of the pain; so far as we have been able to learn, those ulcers which lie directly adjacent to the pyloric sphincter give the quickest pain after a meal. One reason for the pain may be the

acid irritating the raw surface, and another reason may be the existence of a pyloric spasm which squeezes the ulcer and causes the pain. The pain which occurs an hour or two after the taking of food in duodenal ulcer is exactly comparable to the pain which occurs in fissure *in ano* after a bowel movement. The latter patients do not have pain when the feces pass out; the pain comes from the tenesmus after the bowel movement is over. So when food is passing out of the stomach there is no particular pain so long as the pylorus is wide open to permit the passage of the food, but as soon as the food has passed out and the pylorus contracts then pyloric tenesmus occurs and the pain begins. Duodenal ulcers have an exceedingly slight tendency to be latent, whereas gastric ulcers have quite a tendency in that direction, especially when situated at some distance from the pylorus. The nearer the gastric ulcer is to the pylorus, the less the tendency to latency, and the further away, the greater the tendency. So in a case like this, in which the three hemorrhages have occurred at wide intervals, and where there has been no pain or distress between these attacks, we would say from the latency that in all probability the ulcer must lie in the stomach itself and probably at some distance from the pylorus, and that, since it has not been attended with vomiting, it is likely that it does not lie on the lesser curvature. I believe the cases I have seen with ulcer on the lesser curvature have a much greater tendency to vomit than those which are on the greater curvature. Ulcers in the latter locality are very frequently latent both as to pain and vomiting. Even carcinoma of the greater curvature may run an almost latent course until very far advanced. So I think we may almost conclude that, so far as this case is concerned, there is no ulcer in or near the pylorus, and that it is likely to be on the anterior or posterior wall at or near the fundus or along the greater curvature.

I have already said that the passing of blood in the stools and the vomiting of blood permit us only a slight presumptive conclusion. Because most of the blood came from the bowel we cannot say that it indicates necessarily a duodenal ulcer, and we cannot say that hemorrhage by mouth from the stomach indicates a gastric ulcer. Why? Because in duodenal ulcer the blood gets

into the stomach and in turn comes out by way of the mouth, and in gastric ulcer some of the blood passes into the bowel and passes out of the rectum.

There is another thing which I think is true, and that is, that those cases which have repeated hemorrhages are more apt to be gastric than duodenal. The majority of duodenal ulcers do not suffer from repeated hemorrhages from mouth and bowel; most of them have but one or two hemorrhages. On the other hand, gastric ulcers are very apt to recur, and the history of the three attacks and the history of the attacks in this patient's case is rather more characteristic of gastric than duodenal ulcer.

The urinalysis in this case shows a specific gravity of 1020, no albumin or sugar, no red blood-cells or casts. A blood examination on the 17th recorded 2,912,000 red cells and 40 to 50 per cent. hemoglobin. Another examination on the 22d gave 50 to 60 per cent. of hemoglobin, 3,140,000 red cells, 5000 leukocytes, and a good coagulation time—two minutes. The blood-pressure was 145 mm. systolic, 121 mm. diastolic. As to the differential white cell count, the polymorphonuclears were 61 per cent., lymphocytes 38 per cent., eosinophils 1 per cent., so that he had an increase of lymphocytes and a slight decrease of polymorphonuclear cells.

There is an interesting thing about these cases of intense secondary anemia and their effect upon the heart which is exemplified in this case. His systolic blood-pressure is 145, his diastolic 121; the difference between these two is his pulse-pressure, which is only 24. A low pulse-pressure often means a weak ventricle. His pulse-pressure divided by his diastolic pressure gives the heart-load; in his case it is $\frac{24}{121}$, which is remarkably light—20 per cent. It should be 50 per cent. to be normal. In pernicious anemia the heart-load is usually heavy; in pernicious anemia the blood-pressure usually runs about 100 mm. systolic, about 50 mm. diastolic; the pulse-pressure, therefore, is about 50 and the heart-load is $\frac{50}{50}$, or 100 per cent. No wonder that the heart weakens in pernicious anemia, and that edema of the feet and ankles appears. On the other hand, in healthy individuals the systolic pressure measures 120, the diastolic 80, the pulse-pressure 120 to 80 or 40, and the heart-load is $\frac{40}{80}$, or 50 per cent.

Bear in mind the variation in the heart-load in secondary anemia, pernicious anemia, and health.

Taking this man's blood analysis into consideration, we find that his reds are 3,140,000 and the hemoglobin is put down as 50 to 60 per cent., but I rather imagine that the 60 per cent. is a little high in his case. To figure out the amount of hemoglobin he ought to have it is only necessary to make this simple proportion: 3,140,000 red cells is to 5,500,000 red cells as x per cent. of hemoglobin is to 100 per cent. of hemoglobin. That is expressed as a proportion, 3,140,000 : 5,500,000 : : x : 100. Multiplying the means by the extremes, we find that 5,500,000 x = 314,000,000, or x = 57 per cent. In other words, the amount of hemoglobin ought to be 57 per cent., and the amount of hemoglobin which he has is put down as 50 to 60 per cent., so if this analysis is correct his color index would equal approximately unity. This analysis, therefore, means that he is suffering from secondary anemia, a conclusion further strengthened by the absence of nucleated red cells and of anisocytosis—irregularity in the size of the red blood-corpuscles.

Aside from these two facts—his pulse-pressure and the blood analysis—we have no further data. He has no tender spots anywhere, so we are compelled in a case like this to say that the localization, as nearly as can be determined, is presumptively gastric, because it is associated with repeated hemorrhages. The patient being a ward patient no skiagram was made.

Treatment.—If we believed that this man had a duodenal ulcer would it not be wise to advise a gastrojejunostomy? If we believed that he had a gastric ulcer would it be wise to advise a gastrojejunostomy? We must come to a conclusion, because the operative treatment in the two conditions may be diametrically opposite. We would not send such a case to the surgeon if we believed it to be a gastric ulcer, because an operation will not help such a case. The only thing that can help this patient is a rather slow and laborious treatment by diet, which ought to be carried out very carefully with continued examination of the stools for blood. Our practice is to feed by rectum as long as blood is found in the feces. When blood ceases to be present then we give

food by mouth. If the case is not bleeding when we first see it, we give food by mouth; if it is bleeding, we give it by rectum until the bleeding stops, as determined by daily examinations of the feces for blood. The first food given by mouth is usually malted milk, buttermilk, or milk which will not curdle (peptonized). No fresh milk is given in these cases, for it makes hard curds in the stomach, and patients do not do nearly so well as when given malted milk or peptonized milk. Some cases do not take malted milk well because of the sugar in it, but they may do well upon peptonized milk. It is our custom to keep them on peptonized milk for several days and then to add some of the different forms of starch. I have always been partial to arrowroot starch because it is so easily digested. You can subsequently turn from arrowroot starch to cornstarch and then to the cereals, giving the patient plenty to eat, but not much at a time. It is well to have the feedings small in amount and to give them at two-hour or three-hour intervals. After a while, as the irritation becomes less, you can increase the amount you give them, but every patient with gastric ulcer should be thoroughly treated for a period of six months after all signs of trouble have disappeared, because experience shows that an ulcer is not healed the moment it ceases to produce symptoms. This case proves the truth of the statement first made, because when he developed these hemorrhages—he could not develop a hemorrhage without first developing an ulcer—an ulcer must have been already present at least for several days before the hemorrhage occurred.

VISITOR: Would you allow light work while the patient is undergoing treatment?

DR. MIX: I would allow no work at all. The ideal treatment would be for him to go to bed and stay there for six weeks. If these patients would lie down and give up to the treatment they would do much better.

(NOTE.—The patient remained in the hospital six weeks, four of which were spent in bed. He was treated by frequent feedings, at first of milk alone, and later starches were added. He regained his blood rapidly without special medication. When he left the hospital his health had been restored to him.)

DUODENAL ULCER AND COMPLICATIONS

Long History of Abdominal Pain. Hemorrhages with Tarry Stools. Significance of Vomiting of Bile. Prognosis and Therapy.

THIS man has had a lot of sickness; his color is much like the preceding gentleman's, and is due to the same cause—a hemorrhage—but you will notice a very marked difference in the two histories. These cases illustrate very nicely the symptomatology of ulcer of the duodenum and ulcer of the stomach, for this is a typical case of ulcer of the duodenum. You will notice, however, that the symptomatology is very complex, that it is too rich in its flora for a simple duodenal ulcer, and I think you will decide, as I have, that there must be something besides ulcer of the duodenum in his case.

For the last eighteen years, since 1897 or 1898, this patient has had pain in the upper abdomen. It appeared from two to two and a half hours after meals—coming late. At first it usually came on only after breakfast and lunch, not after supper. One would feel that there might be a relationship between work and the delayed onset of the pain after meals in duodenal ulcer, and I believe there is. Presumably, too, there is some relationship between the erect posture and the time of the pain, the erect posture hastening the appearance of the pain. For several years the pain continued, but not constantly, sometimes being absent for a longer or shorter period, and then returning again. He did not notice any exacerbation of the symptoms in the spring or fall during any year.

About five years ago he also began to have a third onset of pain at about 9 o'clock at night. It was dull and gnawing in character, and was relieved by sodium bicarbonate, by food, and by pressure on the abdomen. This latter point is rather interesting because it indicates a sort of colicky pain. Pressure alone on the abdomen irritates an ulcer, but relieves a colic. There-

fore one would imagine that there had been added to the symptomatology of duodenal ulcer a mild degree of pyloric spasm

One thing more which I did not mention—notice how well nourished this patient is, and recall that the patient with gastric ulcer was not nearly so well nourished. The duodenal ulcer patient is usually well nourished, for he has a tendency to eat because food relieves the pain. The gastric ulcer patient is not nearly so well nourished usually because eating does not relieve his pain. He is more apt to fluctuate widely in his weight, going down rapidly when the ulcer is at its height and coming up rapidly when the ulcer is at rest.

Occasionally—four or five times—during these eighteen years this man has vomited a few ounces of greenish, slimy material; he never vomited blood.

(To Patient): Have you vomited this material within the last few years?

PATIENT: Last Wednesday was the last time, and I always vomited when the pain was most severe.

DR. MIX: When was the first of these spells?

PATIENT: Probably seven or eight years ago.

DR. MIX: His first pain began eighteen years ago, and for eleven years it remained much the same, but about seven years ago there appeared a vomiting of bile. This means a reflex anastalsis, and a reflex anastalsis is usually associated with a peritonitic inflammation. I say this because a patient with pure duodenal ulcer never vomits, and for eleven years this man never did; seven years ago a new element was injected into his case, namely, a cholecystitis; and five years ago a colicky spasm appeared for the first time.

Turning to the *etiology of diseases of the gall-bladder*, we find the following causes: first, typhoid fever; second, appendicitis; third, gastric ulcer; and fourth, duodenal ulcer. In the female puerperal fever is a cause, but in the male there are the four great causes just mentioned, and, given a case of duodenal ulcer which has lasted eighteen years, it is almost positive that in the later years there will be added signs of cholecystitis or pericholecystitis, or involvement of the peritoneum of the upper right abdomen.

To return to the further symptoms of his case: After meals he would occasionally suffer gas formation, which would make his clothes uncomfortable, but he had no eructations. His appetite is fair; he eats everything. His bowels are markedly constipated and move only with a cathartic. A patient with cholecystitis without gastric or duodenal ulcer is not constipated; the gall-bladder cases usually have fairly regular bowel movements, but if they are combined with gastric or duodenal ulcer they show constipation. The first patient had a gastric ulcer with constipation, and this man has a duodenal ulcer with constipation. The constipation can be used as a point in differential diagnosis between cholecystitis and gastric or duodenal ulcer.

For a period of a year and a half in 1914 and 1915 this man had no pain, had a good appetite, and remained perfectly well. On July 10, 1915, after carrying a piece of ice upstairs, he noticed that his bowels were going to move and he started to the toilet. When almost there he became weak and dizzy and fell to the floor. He did not lose consciousness, and after lying on the floor for a few minutes he succeeded in reaching the toilet. He was covered with a profuse cold sweat; the bowels moved at this time, but he does not remember the character of the stools. They may have been unchanged at that time because his hemorrhage was then taking place, being caused by the physical exertion of carrying the load of ice. I once had a patient with a gastric ulcer who showed the effect of physical exertion upon an ulcer. His family were moving on a certain day; he was helping to carry heavy pieces of furniture when he suddenly became faint. At the time it was said he was overcome by this work. Then marked pallor supervened. The history clearly showed a hemorrhage brought on by carrying heavy pieces of furniture, due to a rupture of a vessel in the ulcer. With treatment this patient gained about 50 pounds in weight; he was very thin, weighing only 127 pounds; he now weighs 177.

So this patient at the time of his hemorrhage felt very weak, dizzy, slightly nauseated, and was covered with a profuse cold sweat. He went to bed and remained there until Monday—two days. During this period he had no nausea, he did not vomit, and

he had no pain. He then went back to work for the rest of that week, but became very short of breath, especially after slight exertion, and so weak that he decided to stop work and go to the country.

During that week or shortly afterward he noticed that his stools were black; he says they looked like huckleberry jam. They remained that way for about a week; he declares he never passed any blood, but he tells us of the "huckleberry jam"—what better description of a bloody stool do we want? These patients very often give convincing descriptions of their trouble. One patient who came into the ward, a man who had been in this hospital previously and had his gall-bladder drained to relieve him of his pain, when asked if he had ever had any peculiar looking bowel movements, immediately sat up and took notice and said, "Yes." He was an Irishman, unusually devout. He got up early one Sunday morning to go to mass. All at once he felt that his bowels had to move and he started to the bathroom. On the way he felt weak and fell to the floor. After this he had a tarry-black bowel movement, was in bed for several days, and then went back to work. I asked him if he had ever told this to any other physician, and he said he had not; he had never thought it amounted to anything or had anything to do with the other symptoms. If a careful history had been taken as to possible hemorrhage from the bowels his gall-bladder would never have been operated upon.

This man's history gives us the absolute history of a hemorrhage. The first attack was on July 10, 1915; a second attack occurred November 6, 1915. On this date he was taken with the old pain in the abdomen and it has been present almost every day since. On November 7th he noticed that the stools were again black and they remained that way for a week. Last Wednesday, the 17th, he vomited a small amount of a greenish, slimy material which was sour and bitter. This means that the vomitus contained bile. Bile means reversed peristalsis from peritonitis secondary to ulcer, a pericholecystitis, or else a cholecystitis.

He was operated in 1900 for appendicitis, but the operation

failed to relieve his symptoms. We might conclude that this operation was done for the symptoms above, but I found on cross-examination that he had a very bad appendix, that he had an abscess, and a scar from a drainage-tube. Thus we have the thought that there may be a double cause for the gall-bladder symptoms in this case. We do not know which came first; his history of duodenal ulcer began in 1897, his appendicitis came in 1900, but it is probable that he had an appendicitis before his operation.

On January 1, 1901, fifteen days after the operation for appendicitis, he had a severe pain in his chest which breathing and pressure aggravated, and three days afterward he spat up blood from his lung, which relieved his pain. That was a pulmonary infarct, but it was not severe enough to produce a fatal result. It came on very suddenly fifteen days after his operation and was a metastatic affair. It produced a sudden, severe, agonizing pain which lasted three days. He might have lost his life from this pulmonary infarct, but he recovered. The stuff that is spat up after such an infarct is a little round mass, very dark because of the clot of blood in it, and very compact because of the large amount of thick, tenacious mucus of which it consists. If you drop it in water it will sink.

This patient had, in addition, the usual diseases of childhood. About ten years ago he had a discharge from his nose preceded by severe frontal headache; apparently disease of the frontal sinus. He takes cold very easily; he denies venereal disease. He has been married twenty-five years and has no children; his wife may have had one miscarriage. His mother died, presumably of asthma, at fifty-two.

Diagnosis.—Now what ails him? The early part of his history is very plain. It starts out eighteen years ago with an ordinary history of duodenal ulcer. He has: first, pain; second, the pain appears about an hour to two and one-half hours after eating; third, pain is relieved by food; fourth, there is constipation and tenderness over the duodenal region. This is practically the entire symptomatology of duodenal ulcer. Negatively, there was no vomiting, at first no sign of infection in the way of fever,

chills, or leukocytosis; these latter symptoms appear in gall-bladder infections. Fifteen years ago he developed the appendicitis with the formation of an abscess. Eleven years ago some other symptoms were added, namely, vomiting, the vomiting of bile, and a colicky spasm. These are the signs of irritation of the peritoneum, and the vomiting of bile is merely a reflex peristaltic response to that irritation. The patient vomits bile only when he has a peritonitic disturbance—a pericholecystitis, periduodenitis, cholecystitis, or cholelithiasis.

If we examine this man we find that he has tenderness over his gall-bladder as well as over his duodenum. Has he gall-stones? Of course, we cannot say. All we can say is that there is no history of the pain of passing a gall-stone or of the blockade of the common duct by a stone. But when we have cholecystitis we are justified in saying that stones may or may not be present.

This case contrasts very well with the preceding one. The history of hemorrhage is the same in each, but in other ways they are as different as can be. A blood examination on the 19th revealed 2,049,000 red cells, a hemoglobin of 30 to 40 per cent.; this man means a color-index below unity. There are no nucleated reds, and the white count shows 25 per cent. of lymphocytes and about 70 per cent. of polymorphonuclears.

Treatment.—This man, who has suffered for eighteen years, who has had two hemorrhages, and a set of symptoms such as he has, should certainly be operated upon. We would be inconsistent if we said he should not be operated upon, just as we would have been if we had said the other case should be operated upon. Is this the time to operate? No; because he is just over a hemorrhage, and operation should be postponed until the hemoglobin percentage comes up. As soon as this is sufficient to warrant it he will be operated upon. Is gastrojejunostomy the only thing to be done? No; there are, in all probability, periduodenal adhesions which should be taken care of. Many of these adhesions do no harm, but if ever the gall-bladder is bound down in such a way that it is put on tension there is always trouble. If the gall-bladder apex is fastened to the duodenum it is very apt to be pulled upon and to give trouble. Traction adhesions often pro-

duce secondary attacks of vomiting and at operation are to be looked for. And, finally, the gall-bladder must be inspected and drained if necessary.

The **prognosis** in gastrojejunostomy is good after operation. About 85 per cent. of the operations are successful and the percentage is rising. In only a few cases of duodenal ulcer does a vicious cycle appear.

Should his pylorus be closed? The presumption in this case is no. His pylorus is probably not involved, for the reason that the nearer the ulcer to the pylorus the quicker the onset of the pain. His pain usually occurs two and one-half hours after meals, and the presumption is that the ulcer lies at least an inch away from the pylorus. There is not in his case any sign of any scarring of the pylorus, no retention, no stenosis, and that is again presumptive evidence that after eighteen years' duration the ulcer does not involve the pylorus itself.

Medicine will not do this man any good now. If he had come to us when the symptoms appeared eighteen years ago we would not have advised surgical measures, because many of these patients will get well by medical treatment if it is instituted early. If you give them the usual treatment early the duodenal ulcer will heal. This consists of six feedings a day and one in the night if there is pain. They should have three main meals and something in the middle of the forenoon, the middle of the afternoon, and on retiring. At first a milk-and-cream and egg diet is best, but later on the diet list can be quite diversified. The patient should drink plenty of alkaline water; I have these patients put one-half teaspoonful of soda in every glass of water they drink. They are always constipated, and the milk of magnesia or the heavy oxid of magnesia usually effects relief. Will relapses occur? Surely they will. Do relapses occur after the surgical treatment? They do; nearly as often as after medical treatment. You ought to tell your patients that if relapses occur the disease itself is alone to blame. They are to be careful of their diet all the rest of their lives. Don't give too rosy a prognosis—that patients are to be cured for always—because you may not succeed.

DUODENAL ULCER: ATTEMPT AT PERFORATION INTO THE GALL-BLADDER FOLLOWED BY GASTROJEJUNOSTOMY AND RECOVERY

January 18, 1916.

THIS gentleman is a private patient from ———, but he is willing to have his case discussed with you. He was taken ill while on the train to Washington and had to be brought to the hospital. His history is rather interesting, and as the written history is not here we shall have to work it out by question and answer.

DR. MIX: How far back does your illness extend?

PATIENT: About sixteen years.

DR. MIX: He is now fifty-two years of age, so the illness dates back to about the time he was thirty-six.

(To Patient): What was the first thing you noticed? What were your early symptoms?

PATIENT: My trouble came on from overwork; I have always led a sedentary life. Before I was elected Secretary of State I had been a member of the faculty of the University of ——— and of the high school, and after school hours I tried to edit a newspaper; I kept this up for five years, when my health broke down.

When I was made Secretary of State and went to the capital I was completely run down. I would have weekly very bad headaches, which came after excessive eating, or after I had eaten something which contained grease or something that was not well cooked. These severe headaches came on at night, as a rule, and would last until I would relieve them by forcing myself to vomit by running my finger down my throat or by a very strong physic. When under the influence of one of those attacks I felt very, very melancholy; so much so that it would hurt me to have people talk to me or for me to talk to people, but it never occurred to me that my stomach was in the condition it was found to be when I came to this hospital.

The doctors in whose care I had placed myself before, and there were many of them, had always given me something for chronic indigestion, such as cascara, but that would only relieve the trouble temporarily. *The first really hard attack* I suffered was about two months ago, when before breakfast I felt dizzy, and while eating my breakfast I felt more so. After having finished my meal I got up and started to go back into my room, and as I was entering the door I fell to the floor unconscious and remained so for probably a minute. This fainting spell was followed by a very severe headache, perhaps the most severe I ever had. I called in a physician, and he gave me magnesia water and this cleaned me out, but in the several stools that I had for the two days following the fecal matter was as black as tar and as sticky as tar. The doctor made a diagnosis of the case, but I have forgotten what he said it was. But when I returned to ——— I reported to my home doctor and he gave me some medicine for my liver.

On the 21st of December I left home on my way to Washington, in answer to an invitation by the Department of State to act as one of the official interpreters of the International Congress. When I got to ——— I was feeling perfectly well; I was in the company of two friends of mine and was taking the evening meal; I had just about half finished the bowl of broth I had ordered when I felt a very severe pain in the pit of my stomach, a little to the right. That pain would shoot up into the right shoulder-blade just like streaks of lightning and was most excruciating. My friends took me back into the Pullman and administered some brandy and hot water to me. That relieved my suffering for perhaps five minutes, and then it came on for the second time, and was then most severe from the pit of my stomach a little to the right, going up to the right shoulder-blade. While I was thus in pain my breath would be almost choked off; it was with great difficulty that I could breathe.

My friends, seeing the horrible shape that I was in, looked for a doctor on the train, and not finding any, telegraphed for a doctor to get on at ———; I was almost out of my head by that time and so exhausted that I could hardly utter any more cries. The doc-

tor did perhaps the only thing that could be done, which was to give me a hypodermic, and that kept me asleep until I got to Chicago the following morning. At the suggestion of some friends I was brought to Mercy Hospital for treatment.

DR. MIX: And it didn't take as long as fifteen minutes to make the diagnosis. It is fine to have a patient who has the intelligence to state a case; you can't get a better story than this. The story usually comes with a lot of things that have no bearing on the case, but here it is wonderfully simple and wonderfully exact. But there are one or two points that should be gone into to clarify his case. We have now almost everything we want for a perfect diagnosis; we do not, however, have the early history of the case, and the first question I want to ask is, Whether in the early onset of the trouble there was much pain, and if so, when the pain came with reference to meals, and what was the relationship between the pain and the taking of food?

PATIENT: I don't remember ever having suffered any pain before.

DR. MIX: The first pain you ever experienced was the attack of two months ago when you had the attack of vertigo before breakfast?

PATIENT: Yes, sir.

DR. MIX: Was there a time when the taking of food relieved a sense of discomfort or pain, or aggravated it?

PATIENT: Yes, I had that.

DR. MIX: Please tell us about that preliminary distress and its relation to food.

PATIENT: I could always tell when an attack of indigestion was coming on by a distressed feeling after eating, a feeling of fulness and nausea.

DR. MIX: When it came on, would the taking of food relieve the gastric distress or aggravate it?

PATIENT: It would relieve it.

DR. MIX: Did you feel better in some of the early stages when food was in your stomach or when the stomach was empty?

PATIENT: I felt better always when my stomach had food in it.

DR. MIX: Now as to another point; constipation?

PATIENT: Constipation has been my nightmare for twenty years.

DR. MIX: When you would get an attack of indigestion, how long would it last and how long was the period of freedom?

PATIENT: An attack seldom lasted longer than a week; I usually made it disappear in the course of two or three days by taking physic. In the last five years the attacks would come at intervals of a week; sometimes I would extend this period by being extremely careful in my diet.

DR. MIX: You have omitted one other very important factor, and that is the matter of infection.

PATIENT: In the last six years of my trouble I invariably suffered an attack of very severe sciatic rheumatism, mostly in the left leg, and those pains in the past two years have been very, very severe when they came on after the attacks of indigestion.

DR. MIX: As I understood, you were told by your physician that the sciatic nerve should be stretched and treated in various ways, but you always refused to have that done because—

PATIENT: I made a diagnosis of my own case, and could never believe that the pains were really what they call sciatica, but that the attacks were due to an infection caused by the attacks of indigestion.

DR. MIX: What was your reason for that conclusion?

PATIENT: Because these attacks almost always came after an attack of indigestion.

DR. MIX: So he very wisely refused to have his "leg pulled," the nerves stretched, or other things done to it.

PATIENT: About three or four months ago one of my home doctors, when I went to him for treatment for these sciatic pains, started to give me injections of strychnin into the nerves to give them strength for resistance. He can't to this day explain to himself why the strychnin had such an effect as it did; it caused me to vomit until I couldn't vomit any more, and this was followed by a most severe headache.

DR. MIX: Now putting together these facts in the diagnosis, they are: first, evidences of long-continued stomach trouble of

some sort; an indigestion associated with a very severe constipation; a feeling of improvement when there was food in the stomach, followed in the course of time by infectious headaches and sciatica. The signs of the infection were manifest only in the headache and sciatic neuritis; the joints were always free. Then, in addition to that, there is the element of periodicity in the early part of the case, the periods of the attacks being comparatively short and the intervals between attacks comparatively short.

Besides these things, there is another matter of considerable importance, and that is a neurasthenic state. I dare say many physicians who previously looked at him thought he was suffering from gastric neurasthenia. Because of the inability to come to any good diagnosis refuge is taken in "gastric neurasthenia."

The whole case becomes very clear when you analyze his story. The headaches were associated with a feeling of great depression and melancholia, in his case the headaches probably being due to a focus of infection in the gall-bladder. Beginning with his severe attack two months ago was the sudden appearance of the hemorrhage before his breakfast, as proved by the fainting, followed by the tarry stools, denoting hemorrhage from the bowel. He had the dizziness before he went to breakfast, and while he was eating his breakfast this dizziness grew worse, and when he started back to his room he fell in a faint. Evidently during that time he lost a lot of blood, and I dare say his temperature was subnormal.

DR. MIX (to Patient): Did you sweat at that time?

PATIENT: If I remember correctly I did.

DR. MIX: This was followed by the symptoms which always follow such a hemorrhage: the feces were black as tar and as sticky and very foul smelling. One can always tell such a stool from a bismuth stool, which is not sticky. This lasted in his case for a period of about two days and then disappeared. In other words, the hemorrhage was over, but it left him very pale and very weak. He then recovered in a measure from the hemorrhage which took place from an ulcer either in the duodenum or the stomach. All of the blood came from the bowel, and the assumption is that the ulcer is near the pylorus on the duodenal side. The presence of the acute pain and the neurasthenic state are all more in favor

of the duodenal ulcer than of a gastric ulcer, and the hemorrhage itself is rather suggestive, although not pathognomonic, of a duodenal ulcer.

But there is another point which is much more important in the localization of his trouble than the hemorrhage and the preliminary symptoms he had, and that is the second attack which took place on the dining car at ———. This was an exceedingly severe attack of abdominal pain. There was no vomiting as there frequently is in these cases of extreme abdominal pain. The pain had two other elements: one was interference with breathing and the other was pain in the right shoulder; and these things mean *phrenic nerve neuralgia*, and this is almost invariably an indication of an associated gall-bladder disease.

Now the question comes, Was there an attempt at perforation? Our conclusion was that there was such an attempt, and that in this attempt there was enough peritonitis to produce an acute pericholecystitis, which produced the pain in the right shoulder. There was no icterus and nothing to indicate the existence of a stone, unless you look upon this particular attack as an attempt to pass a stone, but, inasmuch as this attack of excessive pain was associated with the attack of hemorrhage two months before, the diagnosis of an attempt at perforation is by all odds more logical than a diagnosis of an attempt to pass a stone. The very severity of the pain is, I think, on the whole, more suggestive of a peritoneal perforation than of biliary colic. There are cases of perforation where patients have been taken ill on a street car, taken out and rushed to a hospital, and operated for perforation of the appendix, when it was really perforation of an ulcer. In each of these cases there was the history of duodenal ulcer over a period of time followed by attempt at perforation.

Was it an attempt at a retroperitoneal perforation? That would not explain the phrenic nerve pain. I think we must conclude that it was an attempt at an anterior perforation, and had this taken place he would have had, of course, an abdomen filled with a load of material and probably would have lost his life, but the perforation did not take place doubtless because of peritonitic adhesions.

We therefore concluded that he had a duodenal ulcer; that there had been a hemorrhage from it two months ago; that it had attempted to perforate, and in this attempt at perforation had involved the gall-bladder; and that there was a marked cholecystitis. It was evident that it was necessary to do a gastrojejunostomy, and he very wisely consented.

How are the headaches and sciatica to be explained? Evidently for a long time past there has been a focus of infection in the gall-bladder sufficient to give rise to these infectious headaches and attacks of sciatica, so we looked upon them as the evidence of an old chronic cholecystitis.

That is as far as we went, but we could have gone a little farther and made an exact diagnosis of the pathway of the attempt at perforation. From the history it would have been evident that the ulcer attempted to perforate into the gall-bladder itself. When the abdomen was opened there was the bleeding ulcer (we had found blood in the stools after he came in and hyperacidity had been found previously). The ulcer was in the duodenum, on that part of the duodenum which was turned toward the gall-bladder, and the gall-bladder was adherent to the duodenum; the ulcer had actually tried to perforate into the gall-bladder. We could have made this out if we had been more painstaking in our analysis of the course of the symptoms. So much of an involvement was there between the gall-bladder and the duodenum that Dr. John B. Murphy, who performed the operation, did not separate the two, but reinforced nature by suturing the gall-bladder to the duodenum in order that the sealing off of the ulcer would be more perfect. Then a gastrojejunostomy was done.

When this patient came in he was in bad condition. He had had a very severe attack of pain and an attempt at perforation, but he can tell you now how much better he feels. He has had very little treatment since the time of operation. The button was used, and he has had some slight attacks of colicky pain as the button has not yet been passed out. There was some ureteral irritation, but that has practically subsided. Of course, he still has his adhesions between the gall-bladder and the duodenum, and they are tighter than they were before. That may give him

some traction pains, but, again, they may not, and they will not interfere in any way with his health. He may experience some little traction for a time until the adhesions have been sufficiently stretched to make the abdominal contents comfortable.

We feel that the ulcer is already healed. He is now taking food by mouth and is almost restored to health. Here, again, is a case of a long-drawn-out duodenal ulcer, going on for sixteen years, going on past a hemorrhage which was not diagnosed, and going on through an attempt at perforation which also was not diagnosed. Examination of the blood at entrance showed 3,680,000 reds, the result of the hemorrhage. An examination of the feces on the 24th revealed the presence of blood, and on the 28th there was still a trace, but on the 29th no blood was present. Before he was operated upon we got rid of the blood by a milk diet and his blood-corpuscles had risen until they were 4,160,000. He was then operated upon, and on the last examination they were 4,200,000. The urinalysis has always been normal.

The case is extremely instructive because it is such a beautiful story of a morbid process told by a most intelligent and careful observer.

PYLORIC STENOSIS AND CHOLELITHIASIS

Frequent Association of Stomach and Gall-bladder Disease. Saddle Ulcer at the Pylorus Causing Obstruction. Operation: Pyloroplasty and Cholecystectomy. Recovery.

October 2, 1916.

THE patient, a married woman aged forty years, entered the hospital June 22, 1916, complaining of pain in her stomach coming on three hours after meals.

Family History.—Her father died at sixty-five years of age of heart disease. Her mother is living and well. She has three brothers living and well, but no sisters. There is no tuberculosis in the family history. A maternal aunt died of carcinoma.

Menstrual History.—Her menses began at the age of thirteen and have always been regular, occurring usually every three weeks and lasting three or four days. They were usually not painful. Her menstrual periods ceased in October, 1914, when she was thirty-eight years old. Her mother also had an early menopause—in her fortieth year. The patient was married at twenty-seven; she has never been pregnant,

Previous Illnesses.—She had the usual diseases of childhood, including scarlet fever. Eleven years ago she had typhoid fever. She has had trouble with her stomach off and on for the past ten years. Aside from these disturbances she has never been ill.

Present Illness.—She is unable to state exactly when her stomach began to trouble her, but says that it has been bothering her for at least ten years. She says that eight years ago the trouble was so bad that a physician made her fast for three weeks. At that time she had what seems to have been a hemorrhage from the bowel, as evidenced by the very dark bowel movement which occurred at that time independently of the taking of iron or bismuth. Careful examination into the circumstances of this al-

leged hemorrhage makes me certain that it was a true melena. Following the hemorrhage and the treatment by three weeks of fasting she remained well for two years. Then her trouble recurred, and ever since she has had periodic attacks of stomach trouble, generally recurring every four or five months.

She enters the hospital because of very severe vomiting. This vomiting began about six weeks previous to her entrance and was very pronounced. For forty-eight hours she was unable to keep down any food, not even water. During this seizure she had severe abdominal pain which was chiefly epigastric. The pain lasted as long as the vomiting lasted. When the vomiting began she first vomited food, but after that nothing but bile came up. She noticed no blood. Ever since that attack she has been unable to eat anything but liquid food in small quantities. As soon as she attempts to eat a meal the pain comes on about three or four hours after she has finished it and she vomits it up. The vomiting gives her relief. The taking of food does not relieve the pain and disagreeable feeling in her stomach. She describes the pain as occurring chiefly in the epigastrium, although she admits that it projects into the back and goes up between the shoulder-blades. She is also very badly constipated. Of late she has vomited food which she had eaten twelve to sixteen hours before. She is sore over her epigastrium, but there are no tender spots that she has ever been able to locate there. She complains bitterly of belching of gas. Bicarbonate of soda relieves the gas, but does not relieve the pain.

Physical examination shows a woman who is rather spare and who has apparently lost a good deal of weight. She weighed 165 pounds when she was married sixteen years ago. After her marriage she gained about 15 pounds more, but after her stomach trouble began she constantly lost, so that she now weighs only 110 pounds. A good deal of the loss in weight has taken place during the last year, especially since the very severe vomiting spell which came on early in May. The patient's color is fairly good, although she is somewhat pale. There is no trace of icterus in her skin or in her scleræ. Her general appearance is not cachectic.

The *blood examination* shows 80 to 90 per cent. of hemoglobin, 5,190,000 red blood-cells, and 8400 white blood-cells.

The feces have been examined for blood, with negative results.

A test-meal showed free hydrochloric acid, 13 instead of the normal 22, and total acidity 45, instead of the normal 60. There was no blood in the Ewald test-meal, no lactic acid, no Oppler-Boas bacilli. No albumin was found on testing the filtered gastric juice.

Examination of the abdomen shows a very moderate panniculus adiposus, with neither tympany nor sinking in of the abdomen. Over the epigastrium are tender spots in the region of the duodenal area and also beneath the right costal border. She also complains that she is a little sore when one presses the fingers in a little deeply beneath the left costal border, but, on the whole, she seems to be more tender on the right than on the left. Deep pressure over the appendix produces no sensation of pain. She has never had an attack of appendicitis that she is aware of, and at the present time has not a particle of soreness in the right iliac fossa.

Examination of the nervous system shows the knee-jerks and ankle-jerks to be present and normal. The pupils are equal and react promptly to light and in accommodation. There are no sensory disturbances in the lower extremities either in the form of paresthesias or of hypesthesias. In other words, there is absolutely no sign of tabes, so that her attacks clearly are not those of gastric crises. Blood examination shows a negative Wassermann, which also tends to preclude the possibility of any luetic trouble.

Diagnosis.—What is the diagnosis in this case? One symptom which she complains of—namely, gas and the belching of gas—can immediately be demonstrated as having no diagnostic value whatever. All cases of gastric and duodenal ulcer, biliary tract involvement, and chronic appendicitis are associated with the belching of gas. The fact that soda relieves the belching is, therefore, of little or no diagnostic value. The same might be true whether the disturbance was due to ulcer, an infected gall-bladder, or appendicitis.

There is a general diagnostic law which it is well to hold to

in any case, and that is, but one pathologic process ever assails an individual. To be sure, such a pathologic process may have several associated and secondary conditions growing out of the primary one, but the general rule holds. Going back to her history, we are confronted with one landmark, and that is her trouble with her stomach eight years ago associated with a hemorrhage from the bowel, which was cured by three weeks of fasting. The cure lasted for two full years. Such a history is the history of an ulcer either of the stomach or duodenum. She can't remember at the present time the symptoms which she had eight years ago, so she can give us no data by which we can distinguish between a gastric and duodenal ulcer, but we can get some data by following out her history of later years.

Perhaps the most important point in her late history was the rather late onset of pain after eating and the rather direct association between the onset of pain and the taking of food. After every meal within a period of from three to five or six hours the pain began, and lasted until the taking of the next meal. For years the only trouble was the pain, but early in May of this year (1916) vomiting was added.

At this point it might be well to say something in regard to vomiting. In gastric ulcer it is a very frequent occurrence, especially when ulcers are located in the region of the lesser curvature. In ulcers of the fundus it is very infrequent. On the other hand, in duodenal ulcers vomiting is unknown unless the case is complicated by cholecystitis or some involvement of the peritoneum in the upper right abdominal quadrant. Taking these facts, which are borne out by numerous clinical histories, as true, it would follow that the attack of vomiting which she describes as terrific, which lasted for forty-eight hours, could not have been due to a simple, uncomplicated duodenal or gastric ulcer. There must be in her case some involvement of the peritoneum or there must be present an involvement of the gall-bladder with or without stones. Of the two latter possibilities the former is the least probable because usually involvements of the peritoneum produce only occasional attacks of vomiting of a comparatively mild type. Usually when the attacks of vomiting are very terrific

we are dealing with a gastric crisis, with some severe involvement of the gall-bladder, or else with cholelithiasis.

Another fact must be mentioned, and that is, of late she has been vomiting food which she had eaten sixteen or eighteen hours before. But what interpretation can be placed upon such a fact? There is retention of the stomach contents either because of a pyloric spasm or because of an organic constriction of the pylorus. Of these two possibilities, again, only the latter is the tenable one. If there were a pyloric spasm it is inconceivable that it would be so regular in its effects day after day. In cases of retention from pyloric spasm the attack is usually an isolated one, occurring on a particular day and then passing away, not to recur for several days or weeks. In this case, however, we find that the patient has retained in her stomach food eaten many hours before day after day. She knows this so well that of late she has limited herself absolutely to liquid diet.

We now are permitted to make two deductions: One is that she had an ulcer, and the second is that she has a pyloric obstruction. These two deductions being established, it is possible to add to them another, and that is, the ulcer must have been situated at a point where it could cause secondary cicatricial constriction. Evidently the ulcer must lie at or near the pylorus. It is not likely that it is on the gastric side because our experience is that ulcers located on the gastric side of the pylorus have pain after the ingestion of food within half an hour to an hour, whereas ulcers on the duodenal side, as a rule, show pain much later. For this reason, then, we are perfectly justified in concluding that she had an ulcer of the pylorus eight years ago, that this ulcer has produced a good deal of scar-tissue resulting in a considerable closure of the pylorus, and that the scar probably lies exactly on the pylorus. We, therefore, have made the diagnosis of pyloric obstruction from a saddle ulcer.

When you look at her history you will immediately be struck by another fact, and that is, the *pain* of which she complains at the present time radiates to her back and between her shoulder-blades. It can be laid down as an absolute diagnostic law that pain of such a distribution is never due to gastric or duodenal

ulcer. Pain penetrating to the back and appearing between the shoulder-blades is of the phrenic neuralgic type, and invariably means either a perihepatitis, a pericholecystitis, a cholecystitis, or a cholelithiasis. The reason for this is obvious. The phrenic nerve, it is true, is the motor nerve of the diaphragm, but it has in it a large number of sensory filaments, and it also makes connections with the abdominal sympathetic. It also sends off filaments which communicate with the supraclavicular and supra-acromial nerves. When, therefore, there is an inflammation involving the terminal filaments of the phrenic it is not strange that an associated phrenic neuralgia should occur. For this reason we find in most cases of biliary tract involvement the well-known symptom of pain in the right shoulder, or of pain between the shoulder-blades or in the back above the region of the diaphragm. Now, in this particular case we have such a phrenic neuralgia, and, therefore, we are driven to the opinion that this patient is suffering from something more than a saddle ulcer with a pyloric stenosis (the saddle ulcer being healed because of the absence of blood in the feces and stomach contents). That "something more" must be, in her case, gall-bladder disease.

In the light of this conclusion, the attack of terrific vomiting six weeks ago, which lasted for forty-eight hours, with such an intolerance to food that even water was rejected by the stomach, makes it evident that the gall-bladder must be involved. It might be involved by adhesions between the fundus of the gall-bladder and the duodenum with traction upon the gall-bladder. This, however, is not likely, because such traction usually produces a steady set of symptoms, whereas in this case we have had a paroxysmal attack of extreme severity, both as to vomiting and pain. One would deduce, therefore, the conclusion that the patient is suffering from a cholecystitis and possibly a cholelithiasis. We cannot be sure of the latter conclusion because there is no history of biliary colic. She has never been icteric and she has never had acholic stools. In other words, her biliary passages have never been blockaded. Had they been, the diagnosis of cholelithiasis would be justifiable, but since there has been no blockade of bile the only conclusion one can reach is that there

is certainly a cholecystitis, with the possibility of cholelithiasis, such as exists in all cases of chronic inflammation of the gall-bladder.

In this connection one fact is of very great importance. It is said in the history that the vomitus consisted almost entirely of bile. In cases of vomiting due to ulcer of the stomach only food is rejected. As soon as the irritating food is out, the vomiting stops, not to recur until more food is taken. In gall-bladder infections, on the contrary, the vomiting of bile is almost a cardinal symptom. It is apparently due to the reflex anastalsis whereby bile is carried into the stomach. It then acts as an emetic and produces the vomiting. Since bile continues to go into the stomach the vomiting continues also. The vomiting does not stop until the stomach ceases to receive bile. Thus it is that in gall-bladder infections periodic vomiting of bile takes place. It is the same sort of reflex as occurs in sea-sickness and in cerebral tumors, and is due to irritation of the peritoneum of the upper right abdominal quadrant.

Conclusion.—We conclude, therefore, that the pathology from which this woman is suffering consists, first, of a pyloric ulcer which developed ten years ago, which was temporarily cured eight years ago by fasting for three weeks. We believe this ulcer to be situated on the pylorus, and believe that it belongs to the group known as saddle ulcers. We, furthermore, are certain, from the retention of food from sixteen to eighteen hours, that there is a marked contraction of her pylorus from scar-tissue. We do not believe that the ulcer is active because we have been unable to find any trace of blood in either stomach contents or feces. We further believe that the gall-bladder is diseased. We are unable to state whether she is suffering from a cholecystitis alone or from a cholecystitis and cholelithiasis. We are not of the opinion that the gall-bladder is under traction from adhesions. We think, rather, that the gall-bladder is itself diseased. We are inclined to ascribe the involvement of the gall-bladder to the typhoid fever of eleven years ago, although we are willing to admit that the pyloric ulcer of itself is perfectly competent as the cause of the cholecystitis.

We have, therefore, advised the patient that medical treatment will offer her no relief. The pylorus must be opened or a gastrojejunostomy done. The gall-bladder must be opened and drained if possible or it may have to be removed. If stones are present they must lie in the fundus and not in the cystic duct or in the common duct. Usually stones in the latter situations are associated with signs of systemic infection. She would be apt to show chills and febrile attacks. She has had none of these. Stones which lie in the fundus are almost invariably quiescent, and rarely if ever give rise to signs of infection, because the lymphatic supply of the fundus is very poor and toxic absorption is not likely to occur. On the other hand, stones which lie at the opening of the cystic duct from the gall-bladder are very apt to give rise to infection because of the very large lymphatic supply of this point. Stones located here are frequently associated with attacks of angular temperature consisting of a very quick rise, perhaps to 102° or 104° F., with a correspondingly quick fall, the whole attack sometimes lasting only six hours. The same is true of stones located in the common duct, only there is added to the symptomatology in this location the symptom of icterus. For this reason we would say that in this particular instance if stones are present they lie in the fundus of the gall-bladder rather than in the cystic duct or at the mouth of the gall-bladder.

The patient left the hospital when the diagnosis was made at the end of June and has been gone all summer. She has been trying to build up a little, and has succeeded slightly by keeping absolutely to a liquid diet consisting chiefly of milk, malted milk, eggnog, and buttermilk. She entered the hospital again October 1st in order to be operated upon, and I have asked Dr. E. Wyllys Andrews to operate upon her this morning.

Operation.—The incision was made in the midline and the pylorus at once exposed. It showed a large, stellate, whitish scar, situated exactly on the pylorus and leading by its contraction to a marked pyloric obstruction. The scar was a typical saddle scar. The pylorus was incised through the scar longitudinally and sewed transversely. The sutures were covered with a purse-string suture of the peritoneum, and the omentum was tacked over

the purse-string suture. When the operation was completed the lumen was sufficiently large to admit the thumb. Subsequent adhesions hardly seem possible if the omentum remains in place. The gall-bladder was then palpated, and was found to be large and to contain stones. The edge of the liver with the gall-bladder was brought up into the field of operation. The wall of the gall-bladder was thick and whitish and the gall-bladder was very much elongated. It showed a very marked cholecystitis and contained two large stones in the fundus. There were no adhesions to the gall-bladder and only a mild degree of pericholecystitis. The gall-bladder was brought into the field and excised. There were no enlarged glands and no signs of carcinoma, either in the gall-bladder or the pylorus. The operative diagnosis was pyloric scar with pyloric obstruction, cholecystitis, and cholelithiasis. Two stones were found in the fundus of the gall-bladder—one at the extreme end and the other close up to it. The gall-bladder wall over the stones was rather thin, but in front of the stones, toward the cystic duct, it was at least $\frac{1}{3}$ inch thick. The diameter of the gall-bladder was about $\frac{7}{8}$ inch and it was 4 inches long. It looked after removal not unlike a sausage- link, both as to size and shape.

November 10, 1916.

NOTE.—*Postoperative History.*—The patient has done very well since the operation. After the fourth day she was given a few drams of hot water to sip. Following the operation she was immediately put upon rectal injections by the drop method. Her tongue remained moist and her general condition good immediately after the operation. She left the hospital five weeks after entering, weighing 6 pounds more than when she came. She was eating anything within reason that she wanted, and she left the hospital in apparently perfect health.

CLINIC OF DR. ISAAC A. ABT

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL FOR CHILDREN)

DISEASE RESISTANCE IN RELATION TO NUTRITION OF INFANTS

Chemical and Metabolic Factors. Alimentary Disturbances in Relation to Thrush and Other Infections. The Exudative Diathesis. The Relation of Fat Feeding to Resistance and Nutritional Disturbance. Starch Injury. Starvation. Rôle of the Mineral Salts.

October 7, 1916.

I DESIRE to spend a portion of this hour in the discussion of disease conditions as they affect the infantile organism and the resistance which the infant offers to disease processes. We know from experience that disease processes affect infants in different ways; that is, an infant that is strong and well nourished is less severely affected than one that is born feeble or delicate or one who suffers from malnutrition. While we would avoid a discussion of immunity in its widest application, nevertheless we may state that natural immunity is one of the most important functions of the living organism as well as living tissue, which ceases with the death of the body. In this field clinical observation is at the present writing in advance of experimental investigation. The knowledge of the relationship between the chemical composition of the body and disease resistance is fragmentary.

Newborn and young infants are peculiarly predisposed to septic infections. This is due in part to the ease with which loss of continuity of the delicate infant skin and mucous membrane may occur. In addition to this, the young infant has a diminished power to develop antibodies when compared to older chil-

dren and adults. It is a well-known clinical fact that young breast-fed infants not only thrive better, but show a greater resistance to infection than artificially fed babies. Moro has shown that there is an increased immunity conferred on the infant by feeding him breast milk, and that there is also a higher bactericidal power in the blood-stream of breast-fed babies. He showed that this germ-destroying power of the infant's blood at birth corresponds with that of the placental blood. Following a breast feeding there is an increase in the bactericidal properties of the blood, but following an artificial feeding there is a rapid decline.

This characteristic condition is not due to improved nutrition alone, for Moro found that even in weak and delicate breast-fed babies the bactericidal powers were greater than in healthy bottle-fed babies. In these observations, if they are correct, we have the explanation why breast-fed babies offer a greater resistance to infection than those who are artificially fed.

Chemical factors, as already stated, though not well understood, bear a relation to the disease-resisting quality of the infantile organism. Czerny and Keller found that the body of the newborn baby is very rich in water, and they think that this explains in part the slight resistance to disease shown by the young infant; with increasing age the water content decreases proportionately and the resistance to disease is decidedly more marked.

Alimentary disturbances also tend to weaken the infant's resistance. It is a matter of wide clinical experience that in infants with persistent digestive disorders infection not only occurs more easily, but also is likely to be more widely diffused in the body, takes a more virulent course, and is refractory to the processes of repair.

Premature and delicate babies are difficult to nourish and show feeble resistance to disease and infection.

These clinical facts may also be stated with reference to communicable disease in young babies. Breast-fed children are not as susceptible to acute contagious diseases (scarlet fever, measles, diphtheria) as artificially fed babies. In those exceptional cases in which breast-fed babies fall ill with an acute infectious dis-

ease, they are less likely to succumb than those who are hand fed.

Sometimes the best and quickest way to control an acute infection (furunculosis, bronchopneumonia) in an infant is to supply him with breast milk.

Disease resistance may be studied in a young baby who is infected with thrush. If, for example, the parasite which produces thrush is placed in the mouth of a normal newborn baby an infection does not ordinarily occur. Even in those cases where the baby's mouth is not subjected to daily cleansing the parasite does not produce the formation of the typical thrush. We conclude, therefore, that the normal newborn breast-fed baby resists thrush infection. This resistance, however, is lost as soon as a mild alimentary disturbance occurs. In the latter instance the thrush fungus makes fast to the mucous membrane of the mouth and grows luxuriantly. If the thrush remains untreated, and the alimentary disturbance is corrected, the mycotic infection tends to disappear.

Alimentary disturbances occur more commonly in artificially than in naturally fed babies. Thrush occurs where there are alimentary disturbances, hence we are justified in concluding that the resistance against thrush is less in hand-fed babies and more in breast-fed babies.

While in young infants only mild nutritional disturbances are necessary to thrush infection, in older infants the mycosis does not occur unless the alimentary disturbance is pronounced. In still older infants (those over one year of age) the disease does not occur except after a persistent infection where the entire organism suffers as the result of a severe nutritional disturbance.

It follows that the natural resistance against thrush is increased with the advancing age of the child as well as by the nutritive condition; the older the child, the less likely he is to be attacked.

Exudative Diathesis.—Under the caption of exudative diathesis Czerny described a definite and well-recognized clinical type which was formerly classified as scrofulosis. Exudative diathesis represents an hereditary familial constitutional state.

It often occurs in all or nearly all the members of a family. The condition is manifested by a definite group of symptoms. There is a tendency to exudative processes on skin and mucous membranes, with hypertrophy of lymphoid tissue of tonsils and nasopharynx, as well as the lymph-nodes of the neck. Many of the newborn infants affected with this condition fail to gain in weight even at the breast; on the other hand, they may be fat and flabby. Two classes commonly occur: the one is fat and of normal weight; the other is thin, remaining stationary or failing in weight. They commonly have seborrhea of the scalp. The hair stands up brush-like and refuses to lie smoothly on the scalp. They tend to have eczema on the face, though this may spread over the entire body. The patients are usually anemic. The blood, on microscopic examination, shows an eosinophilia. As on the skin, so on the mucous membranes, exudative processes occur, manifested by pharyngitis, tonsillitis, rhinitis, secondary glandular swellings, and middle-ear inflammations. Geographic tongue is a frequent and characteristic symptom.

Not infrequently these infants combine asthmatic attacks with lesions on the skin and mucous membrane already mentioned. It is noteworthy, too, that these infants show a tendency toward dyspeptic diarrheal stools.

We have no definite knowledge as to the pathologic process underlying this constitutional state, though we assume that it consists of a disturbance of the normal chemical processes of the organism, and Czerny thinks that it depends upon a derangement of fat metabolism.

Many infants affected with exudative diathesis fail to prosper at the breast. These are the infants who are weaned from the mother because her breast milk is thought not to be sufficiently nourishing or not adapted to the baby. Perhaps one wet-nurse after another is tried, with similar results, the baby failing to thrive. It is, however, important that these babies should receive some breast milk, even if it be small in quantity, because of the difficulty of maintaining a resistance against infection and against nutritional disturbances with an exclusive artificial diet. It is true, nevertheless, that babies of this class who are fed arti-

ficially on a food which is low in fat and reasonably rich in carbohydrate will do better than those who are fed the usual milk mixtures containing what would ordinarily be considered the normal amount of fat.

It is a matter of experience in these cases that intercurrent infections are more likely to occur if these babies are artificially fed and the infections may take a malignant course. This remains true even if the infants are gaining in weight and are apparently prospering on the artificial diet. These infants should be kept on a mixed feeding, even if the supply of breast milk is small. They may advantageously be given a cereal decoction in addition to the milk as early as the third or fourth month.

Czerny has pointed out that the natural immunity of some of these exudative-diathesis babies is remarkably low, even if they are fed exclusively on the breast. In some extreme cases, even where breast feeding is employed, the weight curve may remain stationary as long as the sixth month. The cause is probably to be found in the frequent parenteral infections to which the baby has been subjected. These are the cases in which both family and physician become impatient, though every well-informed medical man knows that to wean such a baby is nearly equivalent to a death sentence. If an attempt is made to feed such a baby artificially the experiment may result in catastrophe.

The Relation of Fat Feeding to Resistance and Nutritional Disturbance.—If we recall the composition of breat milk we note that it contains considerable fat. Few young babies who are artificially fed can tolerate as high a percentage of fat in the cows' milk mixture as they are receiving from the breast. A normal baby can take a large percentage of fat without harm if it is given as breast milk. Breast-fed or artificially fed babies who can digest and successfully metabolize a large quantity of fat show the highest resistance to infection. It has already been pointed out that those who do not assimilate fat in natural feeding have a diminished resistance, as in cases of exudative diathesis.

In cases of artificially fed babies where the fat is excessive or not assimilated even in small quantities a disturbance of calcium metabolism results. Calcium soaps are formed in the intestine,

and consequently calcium is lost to the body. The result is a calcium demineralization or an alkalipenia. Metabolism experiments have shown that as a result acidosis occurs. In young infants the acidosis occurs easily and may be of sufficient degree to endanger life. The extreme ease with which acidosis is produced by alimentary disturbances in young infants is further explained by the fact that they have a smaller alkali reserve than older children or adults. The derangement produced by fat overfeeding or fat indigestion decreases the normal resistance of the baby by disturbing the physiologic composition of tissue substances and fluids of the body.

An interesting series of experiments on animals are recorded by R. Weigert to show the influence of artificial foods on the composition of the animal body, on the one hand, and the resistance against infections on the other. He injected young pigs with living tubercle bacilli in normal salt solution. They were all fed on the same food three months preceding the injection. This food was continued until their death. One-half of the infected animals were fed human breast milk with linseed oil; the other half with buttermilk containing an abundance of flour and sugar. All of the animals became infected. Those that obtained the fat diet stored up more fat than those that obtained the carbohydrate. Those that were fed carbohydrate became severely ill, more organs were involved by the disease, it became widely and rapidly diffused, and the nutrition of the animals suffered more markedly than those animals who were fed the fat food. Weigert succeeded in showing experimentally, probably for the first time, that the prognosis in tuberculosis and the resistance of the organism against the disease depend in a considerable measure upon the dietary régime of the animal.

Horneman and Thomas, in a series of experiments, showed that by feeding food containing a large quantity of protein the animals exhibited an increased resistance against tuberculous infection.

Starch Injury.—It is a well-recognized point in dietetic management, whether in adults or infants, that a well-balanced régime is necessary for perfect health. A one-sided diet fails to keep the

animal in health and lowers resistance. If infants are fed an exclusive carbohydrate diet, or if they receive excessive quantities, they will ultimately present the condition of starch injury. This may occur as an atrophic or hydremic type.

The *atrophic type* resembles in every respect the condition which is described by Finkelstein as decomposition, though in this instance of another etiology. This class of patients lose excessively in weight, become weak and emaciated, and suffer a great reduction in their normal immunity. The atrophic form occurs more readily in those cases where the food is poor in salt and sugar.

In the *hydremic form* the children make the impression at the outset that they are gaining in weight and progressing unusually well. Very shortly, however, they become edematous and appear like patients suffering from the anasarca of renal origin. The face is swollen, the eyes are closed by edema, and the subcutaneous tissue becomes waterlogged. The urinary examination shows the absence of albumin and sugar as well as the absence of formed elements on microscopic examination.

This type of starch injury is more likely to occur in those infants who receive salt or sugar, or both, added to the starch. A difference of constitution may explain why the atrophic form occurs at one time and the hydremic type at another. We may assume that a certain group of children naturally tend to retain water in the body, while another group have not developed any ability to increase the water content.

The resistance against infection in both types is diminished, though more in the hydremic than in the atrophic form. In the former the increase in water content is considered to be an important factor in the reduction of the normal resistance to infection. Furunculosis, rhinitis, otitis, bronchitis, and pyelitis are a few of the infections which not uncommonly complicate this severe disturbance of nutrition.

Underfeeding.—Starvation diminishes resistance to infection. It has been said that a starving population is rendered more susceptible to typhus fever than one that is normally fed. This statement lacks exact clinical confirmation.

Much more convincing are some experiments by Canalis and Morpurgo. They selected chickens, pigeons, and white rats as the animals for study, and as the organism to be inoculated they chose the anthrax bacillus. Immune animals may sometimes be rendered susceptible if previously starved. Immune pigeons which had been deprived of food for six days were inoculated with anthrax, and shortly after the injection were fed again. They did not acquire the disease. If the food deprivation lasted longer than six days previous to inoculation, they fell ill with the disease. If pigeons are deprived of food for two days after the injection, anthrax will develop. In animals that are starved for several days the intestinal wall becomes more porous and permits bacteria to migrate through its coats. In rabbits that had been starved several days bacteria could be found in the blood and organs. In dogs and guinea-pigs the small bowel is relatively free from bacteria under normal conditions. If the animals have been previously starved, however, the intestinal tract will swarm with micro-organisms.

It is pretty well proved that starvation increases the predisposition to a great variety of infections and renders the course of the same more severe.

Behrend, in a recent publication, maintains that he has frequently observed infants suffering from an insignificant rhinitis or bronchitis, who, if subjected to starvation, became ill in a few days with a severe pneumonia. He supports his statement by the report of a large series of cases.

Prolonged starvation is unphysiologic, tends to cause precipitous loss in weight, lowers the natural resistance of the patient against infection, and diminishes the reparative power of the organism.

Mineral Salts in Relation to Nutrition.—The quantity of iron salts contained in breast milk it is thought is insufficient for the needs of the infant. The newborn baby comes into the world with a reserve supply of iron stored up in its body, so-called "iron depots."

If babies are kept on an exclusive milk diet for an excessively long period anemia may become marked. Children who receive

at the fifth or sixth month additional food, such as fruit juices, beef juices, and vegetables, are supplied with the necessary quantity of iron salts.

That there are other substances than the common food elements which may produce disease is shown by the study of beriberi. We assume that there is some active substance in the shell of the rice kernel whose presence protects the patient who is partaking largely or exclusively of this food. The absence of this substance or vitamin tends to the production of beriberi.

We are considerably at sea concerning the relation of foods to such common nutritional diseases as rickets and scurvy.

To conclude, we feel justified in assuming from clinical data that artificial feeding or nutritional or alimentary disturbances, whether due to artificial or natural feeding of infants, lower the resistance against disease and predispose the baby to secondary infections.

DECOMPOSITION

Presentation of a Typical Case. Etiology, Symptomatology, and Clinical Course. Management in Detail, with Special Reference to Feeding.

October 14, 1916.

THIS poor little emaciated baby that I present to you this morning is eight months old. He has been with us in the hospital for seven days. The early history that relates to his birth and the first days of his life presents nothing of note. He is poorly developed and he has not, up to the present time, sat erect. He has erupted no teeth.

Note the feeding history: He was at the breast only two days. After that he was placed on modified milk, and recently the mother has been giving him a formula consisting of one-half milk and one-half water. Five ounces of this mixture were given to him five times a day, each bottle containing one-half teaspoonful of granulated sugar. Recently he was fed farina once daily, and on the advice of a neighbor lady he was given two soft-boiled eggs daily. Is it considered good practice to give an eight-months-old baby two soft-boiled eggs daily?

We will answer that question by saying that, as a general rule, normal babies are not fed with eggs until after the fourteenth or fifteenth month. Many babies even at this age show an idiosyncrasy against eggs, and this is particularly true of that group of babies that we classify under the caption of "exudative diathesis." A description of this condition was referred to in the preceding lecture, but to think of giving a poor weak baby, such as this one, two eggs daily **is** unphysiologic and beyond all reason.

According to the history, this baby has had diarrhea nearly all summer. He had five to six stools a day which have been greenish in color and contained mucus. For the past two days

the history says he has been vomiting after each feeding. Recently he has had ten to twelve stools a day which have been green in color. The baby sleeps well.

The family history is said to be negative.

If we examine this baby we observe a very thin, emaciated little being, with a skin wrinkled and dry and inelastic. The skin on his face is folded. His features are mask-like and his face looks like that of an old man. He seems very weak and has been crying or whining in a weak manner a great deal until today. He is now very quiet, as you observe.

Examining his head, we notice that he has a small meningocele in the occipital region. The pupil of the left eye is larger than that of the right, wedge shaped, reacts slowly to light. The pupil of the right eye is normal. The fontanel is very much retracted and the sutures are open. The eyes are injected; pus exudes from the left eye, and there is marked conjunctivitis, particularly in this eye. There is desquamation of epithelium from the conjunctiva, which lies slightly adherent over the cornea. The irregular shape of the left pupil and the meningocele are congenital in origin. The mother informs us that this condition was noticed at birth. The baby coughs slightly. The mucous membranes are pale. The tongue is dry. When one puts his finger in the cavity of the mouth one receives the impression that it is cool; lacks the normal warmth. There is no rigidity of the neck. The skin all over the body has lost its elasticity; its normal turgor. It seems tough, like tanned leather. If the skin is stretched it does not snap back like live rubber, but remains in the position in which it has been placed. The muscles of the upper arm seem to be contracted. They are hypertonic, resembling the condition of rigor mortis.

The pulse is feeble. The heart's action, however, is very slow.

Examining the thorax, one hears a few fine crepitant râles posteriorly.

Note the malformation of the chest. The upper portion of the sternum protrudes, and the circumference of the chest in its upper portion is small, while the lower ribs tend to flare. There is no

marked evidence of rickets. The spleen is not palpable. The liver can be felt one or one and one-half fingerbreadths below the costal arch.

Let me call your attention to these purpuric spots covering the surface of the abdomen. We note this condition frequently in this class of babies, and we speak of it as *marantic* or *cachectic purpura.*

Let me emphasize, too, that the urine contains a trace of albumin, many leukocytes, and no casts.

Note the blood examination:

85 per cent. hemoglobin;
4,920,000 reds;
9200 leukocytes;
41 per cent. polymorphonuclears;
48 per cent. mononuclears;
9 per cent. large mononuclears;
2 per cent. transitional.

The throat culture was negative; that is, there were no diphtheria bacilli.

The von Pirquet reaction was also negative.

The baby weighed 7 pounds when it entered the hospital. It has lost an ounce, and now weighs 6 pounds and 15 ounces.

The baby's temperature on entrance was 98° F. The temperature today is 96° F.

The baby has been receiving breast milk ever since its entrance into the hospital.

Our nurses have been taking very zealous care of this baby. We have feared for the baby's life from day to day, and we consider ourselves fortunate that it is still here.

Now let us summarize the findings: A baby eight months old, emaciated beyond power of adequate description, with a temperature of 96° F. and with irregular respiration, lies before us in a semicomatose condition, with its mouth open, eyes staring, its features drawn and fixed. It weighs only 6 pounds and 15 ounces, shows a loss of turgor of the skin and a hypertonic condition of the muscles, and is in such a weak condition that dissolution seems imminent almost any moment.

What is the diagnosis?

A Visitor: Atrophy.

Dr. Abt: The disease has been variously described under the terms "atrophy," "marasmus," and, in the light of more recent classification and more recent thinking, we call it "decomposition."

Etiology.—Let us consider the causes which were at work to produce this illness in this particular baby. We are told that the baby suffered from a long-continued indigestion and diarrhea. The infant has had a diarrhea nearly all summer, and has had five to six stools a day, which were greenish and slimy. For the last two weeks there has been vomiting after each feeding and the baby has been continuously losing in weight. We note that the patient has been subjected to long and continued alimentary insults, and the baby eventually is brought to us in the condition in which you see it—exhausted, atrophied, and, to all appearances, beyond repair.

We note, then, that there has been a repetition of alimentary disorders before this severe atrophic condition has been brought about. Of course, it must be stated that there are other causes than strictly alimentary ones which may produce decomposition or atrophy. For example, a baby who has malformations of the various portions of his body is difficult to feed. This is true of a baby with spina bifida, with severe cleft-palate or hydrocephalus, or sometimes congenital heart disease. The slight cerebral meningocele in this case has probably had no influence on the present condition. Such babies frequently suffer from decomposition either on account of bodily weakness or on account of difficulty in partaking of food. Babies who suffer from either acute or chronic infections—in the first instance a pyelitis, and in the second a tuberculosis—may become markedly atrophic as the result directly or indirectly of the infective process.

A single diarrheal attack seldom results in decomposition, though delicate infants, and those who are prematurely born, are particularly predisposed to dangerous loss in weight and to sudden decomposition. In older infants the summation of attacks is the important factor in the production of the disorder.

Underfeeding plays an important part in the etiology. Those babies who are more or less starved are likely to suffer from decomposition. This condition may occur at the breast or at the bottle. For example, a baby at the breast may receive insufficient food, possibly because the baby is unable to withdraw the milk or the mother is secreting insufficient quantity. At any rate, the baby is receiving much less than it needs. Or, when the baby is fed artificially, the food may be insufficient in quantity or improper in quality.

Very frequently after a baby has had an acute attack of diarrhea normal feeding is resumed with some difficulty, and sometimes the process of withholding the food is continued for a long time, or there may be a number of short and frequently repeated starvation periods. The patient experiences difficulty or inability to recover weight, and, as a matter of fact, long-continued underfeeding or frequently repeated shorter periods of starvation are likely to lead to decomposition. Or a baby that is kept on an unbalanced or one-sided diet may suffer from decomposition. For example, a baby who has suffered from some gastro-intestinal disturbance placed on barley water or barley gruel seems to do fairly well for a time on this diet; it does not increase in weight markedly, nor does it lose, and very frequently the mother gives this food day after day, until finally the baby is no longer able to derive sufficient nutriment for the purpose of growth and development from this scant diet, and he begins to show marked loss in weight. The decomposition in this instance has occurred, it is thought, from an injury produced by the long-continued and overuse of starch.

Symptomatology.—What are the symptoms of decomposition? Note this baby again: The skin is pale, somewhat anemic; the mentality most frequently remains good until near the end. As a rule these babies are bright until they approach the fatal termination. This baby is emaciated to an extreme degree; the fat seems to have disappeared from the body; the skin lies in folds, is inelastic, and its normal turgor has been lost. What do we mean by "turgor"? You will recall that turgor is a botanic term, and in that connection refers to the loss of

water from the structure of the plant, and as a result the plant wilts, loses its fulness and its rotundity; and so in babies the wilting process occurs, the fulness and rotundity disappear, the skin lies in folds, and has lost its snap or its elasticity.

Note this baby's face: The senile appearance, the folded skin, the loss of rounded contour and baby fulness; his face seems angular, the eyes stand out prominently; he looks like a little old man who has been withered by the storms of many winters. His mouth seems very large, and this appears all the more so because of the atrophy. When he opens his mouth to cry the cavity seems huge compared to the small sunken face. The baby's fist can be introduced into this cavity.

The thorax seems small, the ribs appear through the external integument. The abdominal wall is thin, and the muscle substance is so atrophic that the peristaltic movements of the intestine may be observed through the abdominal wall.

The breathing is irregular; the expiration tends to be prolonged, and expiration and inspiration follow one another in an irregular manner. The pulse is usually slow, and in some instances it may be as low as 80, though it may fall to 60.

You will remember that this baby's temperature was 96° F., and that it has been subnormal for a considerable time. Subnormal temperature is an early symptom of decomposition. In cases where the temperature tends to rise materially and continues high an element of alimentary intoxication may occur or is to be feared. Long-continued subnormal temperature, such as 95° or 96° F., indicates a low condition of vitality, and a dangerous degree of collapse may occur at any time.

I have told you that in this case the urine was free from sugar and the slight amount of albumin which was found was due to the presence of pus, which was evidence of a pyelitis, and this applies to the majority of these cases. The kidneys seem to remain, for the most part, normal.

It is a remarkable fact that such babies as the one I show you here this morning retain their appetite and their desire for food. They are extremely thirsty and desire great quantities of water. It is not an uncommon sight to see one of these babies suffering

from decomposition wide awake, mentally active, and overstimulated, throwing himself about from side to side, hands in the air, putting his fists in his mouth, and evincing great relief when given fluids, particularly water. These patients frequently vomit. The appearance of the stools is variable; diarrhea may be more or less constantly present, though in some instances hard, dry soap stools are observed, and at other times undigested or dyspeptic stools, containing particles of food, curds, mucus, and more or less fluid. When the babies are fed considerable fat, undigested stools containing fat are observed. Occasionally the stools are very dark or black, indicating possibly that some blood has found its way into the intestinal tract. Indeed, some of these babies do suffer from intestinal hemorrhage, and it has been found that duodenal ulcers may occur in these young infants; that more or less oozing takes place and, as a result, the bloody stools.

With increasing loss in weight there is a loss of bodily functions, but, possibly more important than anything else, the tolerance for food is lowered and the normal immunity of the body is diminished. When the baby's body is so damaged that his tolerance for food is diminished or lost to such an extent that food of almost any kind, prepared according to our best knowledge, fails to agree with or to nourish the baby, then the life of that individual may be despaired of. Indeed, the baby continues to lose in weight in spite of all attempts of feeding. It has been suggested that a baby who has lost one-third of his body weight is in great danger, and, indeed, his tolerance for food has been so seriously damaged that he is no longer viable. Sometimes a short period of starvation in an already developed case of decomposition produces a precipitous loss in weight and fatal collapse.

I think we have already referred to the fact that these debilitated babies, who have a lowered tolerance for food, who are more or less continuously losing in weight, are very susceptible to infections of various kinds. Bronchitis, furunculosis, pyelitis, grip, and otitis may occur, produce some fever reaction in these infants, and, indeed, not a few of the protracted cases of decomposition die as the result of the parenteral infection. You will

recall that this baby showed considerable pus in his urine, and he is undoubtedly suffering from a terminal pyelitis.

It would be a mistake to permit this desperately sick patient I have shown you this morning to serve as an illustration of every kind of decomposition. The fact is, there are mild cases where the weight remains more or less stationary with slight tendencies to loss, the weight curve is zig-zag in character, and the decomposition may be latent in form. The true nature of the impending atrophy occurs when the baby falls ill with a slight diarrhea, or when there is some decided change in the baby's diet or if an infection occurs, and under these circumstances a latent decomposition may become manifest.

More striking perhaps are the results observed when starvation is instituted for therapeutic purposes. In these cases the loss of weight may be very sudden and very marked. In taking a history of one of these babies the diagnosis is frequently made or suggested by the fact that the baby has suffered from repeated alimentary disturbances, has suffered from mild infections, and has shown loss in weight. Individual symptoms, like intense pallor, subnormal temperature, irregular and slow pulse, are suggestive symptoms. Loss in weight eventually occurs in almost every instance.

Clinical Course.—The course of the disease depends upon the severity of the disturbance. Every case of decomposition offers a great menace to the baby's life. This is particularly true because of the lowered resistance of the infant and the diminished tolerance for food. The diminished tolerance for food implies the great difficulty in feeding these infants; to find the proper food in quality and quantity means to restore the baby; failure to find the proper food implies that starvation is imminent. For this reason catastrophe is always threatening. It is true that the food tolerance can be raised if the proper diet is found. In babies that are markedly reduced it is more difficult to prevent infection. It would be a mistake to think that all babies who suffer from decomposition are irrevocably lost, because many of them under proper diet and nursing may recover in the course of weeks or months. The severe cases are more difficult to reach by

therapeutic measures. Not infrequently they die during the first days in collapse. If these patients survive the early period, life may be saved, or a patient may be on the road to recovery and may be lost as the result of a renewed attack of diarrhea or an infection.

Let us interrupt for a moment to observe the patient before we return him to his bed. Note again the confluent purpuric eruption over his abdomen. We observe this frequently in cases of marasmus, and we designate it as a type of cachectic purpura. It occurs particularly in this location in babies suffering from decomposition in the last stage. I have rarely seen one of these babies with this form of purpura survive.

As we are proceeding with our elucidation of this disease condition we naturally become very curious as to its *nature*, whether the changes consist of degenerative processes in tissues or organs, whether manifest histologic changes are produced, or whether there are perversions of chemical processes. As nearly as we can learn, the most marked changes in the metabolism or the chemistry of the tissues consist in a loss of mineral substance to the body. This results in a *demineralization*. We have learned that the soap stools result in the loss of calcium salts being combined with fatty acid, and even in the diarrheal stools there is a loss of alkali from the body. The increase of the ammonia content of the urine is a secondary phenomenon, and occurs because so much of the calcium used for neutralizing the acids has been lost, consequently ammonia appears secondarily as a neutralizing body.

There are several points about the reaction of these babies to food that are of interest. For instance, if a baby with decomposition is fed for twenty-four hours with weak tea and saccharin the weight curve in the milder cases may show a moderate fall, the temperature, if there has been any increase present, becomes lower, and the pulse tends to become slower. If, however, the case of decomposition is of the severer kind, loss of weight may be very marked; indeed, the severer the type of the case, the more precipitous and the greater is the loss in weight. In a child that is normal or nearly normal after a period of twenty-four hours' starvation loss in weight will be only 3 or possibly 6 ounces,

whereas in a baby suffering from decomposition that has been starved the loss may be twice or three times this amount. These reactions on the part of the pulse, temperature, and weight are sufficient to enable one to make the diagnosis of decomposition.

The **treatment** of cases of decomposition in order to be successful must be so planned that the loss or the great diminution in tolerance for food and the diminished immunity shall be remedied. We may say at the outset, then, that the reconstruction of the functions and tissues of the body and the repair and restoration in the normal processes of development depends upon food supply. We may say in passing it is not a problem of finding a pharmacologic preparation or a vaccine or an antitoxin; it is simply a question of feeding the baby properly. It may be stated at once that the best results are obtained, as a rule, by feeding the baby breast milk. The breast milk restores in a most efficient manner the normal condition of the intestinal epithelium, and also tends to assist more rapidly in the repair of tissues and cells which lie outside of the intestinal lumen. Breast-milk feeding, too, in these cases seems to protect the baby against infection, and we have already learned that these babies succumb rapidly to prolonged starvation, so that in cases where the breast milk is not obtainable other forms of food suited to the baby's digestion must be substituted. Perhaps it may be well to remind you again of the fact that babies suffering from decomposition must not be deluged with the breast milk or given excessive quantities. It is better to give small quantities frequently repeated at the outset. Many a baby who has been furnished breast milk *ad libitum* in the treatment of decomposition has been rendered more acutely ill by overdosage with the breast milk. This must be guarded against by giving the infant small doses, 1 or 2 ounces to start with, eight, nine, or ten times daily. It must be remembered, too, that in the majority of these cases the baby does not proceed to gain at once even if he is fed breast milk. After the baby has been upon this food for several days he grows happier, shows more vigor, the skin shows a better color, it becomes more elastic, the baby tends to become more rotund, the folds disappear from the face, though with all the baby has shown little or no increase in weight. This

period of reconstruction and repair we have learned to know as the *reparation stage*. Physicians as well as nurses of experience will record instances where the wet-nurses have been changed or discharged during this period because the baby failed to gain in weight. We know, however, that this period of reparation may last for days or weeks and is not inconsistent with normal progress; indeed, in most cases it is what we have a right to expect in the way of improvement.

Another word may be mentioned in this connection. Where these babies do not increase in weight at the breast, other foods are frequently added; powdered casein has been recommended to precede each breast feeding, buttermilk mixtures, skimmed milk, or, in some instances, sugar and carbohydrates in small quantities may be added. Great caution should be taken in treating these cases by the addition of carbohydrate or sugar, because experience teaches that these substances in excessive quantity may produce a renewed attack of diarrhea, or, indeed, a decomposition may be transformed into an alimentary intoxication as a result of their administration. It should be emphasized, in this connection, that if the baby is to be treated with artificial food, excess of sugar, carbohydrate, and fat is to be avoided at the beginning of the treatment. Those of us who have tried the albumin or protein milk therapy are convinced of its value in the treatment of these severe alimentary disturbances, particularly in the treatment of decomposition.

In a previous lecture I have already detailed the directions that have been given by Finkelstein and Meyer for making this milk. Briefly, you will remember that it is a food that contains casein slightly in excess, and that the sugar and the salts have been partially diminished by the removal of the whey and the substitution of buttermilk and water. At any rate, after the short period of starvation advised for these cases, the albumin milk, if it is to be used, should be begun in small quantities, 1 ounce in young infants or 2 ounces in somewhat older infants, given at short intervals, so that the baby may be receiving instead of five or six feedings per day eight or nine or even ten feedings. No sugar should be added at first; later on, when the baby's

diarrhea is under control, the sugar may be added, beginning with 2 or 3 per cent. and gradually increasing it to 5 per cent.

The albumin milk is not a permanent diet and may be continued four, five, or six weeks, and then may be substituted by the ordinary milk mixtures again. It must be remembered that in the ordinary albumin milk there is little or no carbohydrate and that without carbohydrate babies will not gain in weight. For this reason it is necessary at an early day to resume carbohydrate feeding by adding the sugar, usually in the form of malt sugar of one kind or another.

Another point in the administration of this food consists in the fact that when the diarrhea has become controlled, and the baby has improved in every direction, the small frequent feedings may be succeeded by larger quantities at longer intervals; that is, a baby may now be fed five or six times per day and given the number of ounces that would be proper for a baby of that age.

I have already said that at the very outset these babies suffering from decomposition require little or no medicine. It is true that symptomatic treatment is sometimes called for in the severe instances of collapse. Where stimulants are required, caffein or camphor may be indicated in hypodermic form, or small doses of alcohol are sometimes indicated, particularly in those cases where a rapidly combustible food is required it seems to be the urgent indication; 10 to 20 drops of whisky or brandy may be repeated every two or three hours. Sometimes other heart tonics, like digitalis or strophanthus, seem indicated.

These babies should be protected against catharsis, as they are in no condition to withstand increased diarrhea or intestinal irritation. It is a question how much good we accomplish by rectal alimentation or the rectal injection of normal salt solution, because these babies retain the fluids very poorly. If a continuous drop method is employed, they absorb the water very slowly and only for a short time. Irritation soon becomes marked and the results are of doubtful value. I have rarely seen results from the subcutaneous infusion of normal salt, though in the desperate cases it may be tried when all else seems to fail. Occasionally when the babies are in collapse results are obtained by placing

them in mustard packs or in mustard baths, so as to restore peripheral circulation. These babies should be kept under covers, and every effort should be made to maintain bodily heat, supply plenty of fluids, and fresh air.

For diarrhea, chalk mixture of the Pharmacopœia may be given in teaspoonful doses every two hours. Sodium bicarbonate may be added where acidosis is present or suspected.

CLINIC OF DR. JAMES T. CASE

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BARIUM DIAGNOSIS

Further Considerations in the Management of Roentgen Studies of the Gastro-intestinal Tract.

Routine Technic of Barium Meal Study of the Esophagus and Stomach Fully Described in a Previous Lecture. The Present Discussion Concerns the X-Ray Study of the Alimentary Tract as a Whole, the Technic being Given for a Routine Gastro-intestinal Study After a Barium Meal.

Graphic Chart Showing Normal Intestinal Motility. Technic and Value of the Opaque Enema. Intestinal Adhesions. Value of Screening in the Horizontal Position.

The Fallacy and Uselessness of the "High" Enema. Method of Clearing the Colon Without Laxatives.

In previous lectures I have outlined the course followed as a routine method in the roentgen investigation of the stomach, and in the last lecture I took up in detail the management of a case to be examined for suspected duodenal ulcer. I also described the method of preparing the patient for examination, together with the other preliminaries, which include the necessary adjustment of the eyes of the observer before he can hope to make satisfactory screen observations.

Today we will consider the technic of the gastro-intestinal roentgen examination, giving special attention to the behavior of the intestine during the passage of the opaque meal along the tract. We have listed for today's study a number of cases from which I have selected one for demonstration of the normal findings. This is a nurse, a young woman who is presumably in good health, who took the barium meal the day before yesterday in the

morning. This consisted of our standard *x*-ray test-meal, which begins with one-third of a glass of water into which has been stirred a level teaspoonful of barium sulphate specially prepared for *x*-ray purposes. This is followed by two glasses of buttermilk, each containing an ounce of barium sulphate.

During the ingestion of this opaque meal the patient was studied by my associate, Dr. Moir, in the manner described in a previous lecture (Medical Clinics of Chicago, Vol. II, No. 1, p. 199). Dr. Moir made a number of tracings and roentgenographic records, so that we will be able to review the written record of his fluoroscopic observations and, for purposes of class work, avail ourselves of a graphic representation of the motor behavior of the alimentary tract, which in this particular patient is about normal, so far as we can judge.

We now see the patient or, rather, the subject for the tenth time, forty-nine or fifty hours having elapsed since the ingestion of the opaque meal. Ordinarily we do not see patients quite so frequently, but in the present instance our object was to secure some graphic charts illustrating various appearances of the opaque meal in its passage along the alimentary tract. At the present hour (the fiftieth) we see that the abdomen presents a uniformly gray shadow, without any barium shadows. This implies that the colon has been entirely emptied of any barium residues resulting from the opaque meal. There are a very few light areas in the hepatic and splenic regions, where small quantities of gas may be noted in the right and left flexures of the colon. We also observe a little gas or air in the fundus of the stomach under the left diaphragm. These gas collections in the colon permit us to make out clearly the lower border of the spleen and liver, and to observe at the same time that both these organs are normal in size and position. The moderate amount of gas in the bowel is probably a normal finding. Excessive quantities of gas in the bowel are evidence of coprostasis and, consequently, such abnormal gas accumulations in the bowel are found in most cases of chronic constipation. By studying the haustral markings the gas accumulations in the colon may be distinguished from similar collections in the stomach or small bowel.

The passage of the opaque meal having been observed from the time of its ingestion until the disappearance of its last trace, the patient is ready to be prepared for the injection of the opaque enema to complete our gastro-intestinal study. We will, therefore, instruct the patient to resume the various treatments which may have been interrupted in order that the *x*-ray observations on the stomach and bowel might be made after the ingestion of the opaque meal. Early tomorrow morning, say at 6 or 7 o'clock, she will receive cleansing enemas to empty the colon thoroughly in preparation for the injection of the opaque clysma under fluoroscopic screen guidance. The opaque enema will be administered two or three hours later, we will say at 9 o'clock.

It is very important that the directions concerning the patient's preparation for the opaque enema examination be followed out minutely. The preparation consists of three cleansing enemas, which are given early in the morning. Three warm enemas of 2 or 3 pints each should be given, each to be retained for a few minutes before expulsion, and each to be fully evacuated before the next one is injected. It is quite unnecessary to give large enemas. The injection should never be so large as to cause the patient any unreasonable or unbearable amount of discomfort, and any such unpleasant symptoms may be avoided if the enemas are injected slowly enough. An ordinary rectal point should be used. It is not only useless but quite unnecessary to employ the colon-tube or to attempt the so-called "high enema." The cleansing procedure being satisfactorily completed, the patient may go to breakfast, after which she may report to the *x*-ray department at the appointed hour, between 9 and 10 o'clock, for the injection of the opaque enema.

This plan of preparation secures a most thorough evacuation of the colon. Long experience has shown that in the majority of cases—in fact, with very few exceptions—the meal of the night before has entirely passed into the colon by the time of rising in the morning; and from the colon it may be dislodged by enemas if they are given in the manner specified above. There must be an interval of at least two hours between the time of administration of the cleansing enemas and the opaque enema in order to

favor absorption or expulsion of whatever residue of water the bowel may retain, and which might seriously "dilute" with the shadow of the opaque enema were it administered immediately after the cleansing procedure. This precaution is not necessary unless a special point is made of the study of the efficiency, or otherwise, of the ileocecal valve. In that case the opaque enema should be given before 10 or 11 o'clock in the morning, since by that time portions of the 8-o'clock breakfast are already beginning to arrive in the terminal ileum, interfering with the passage of the clysma into the ileum through the incompetent valve at the ileocecal orifice, should this valve prove to be inefficient. It is, therefore, quite evident that any test of the competency of the ileocolic valve must be made under circumstances which will insure an empty terminal ileum and cecum prior to the injection of the opaque clysma, and this condition will only obtain during the early morning hours before the head of the breakfast column begins to arrive in the terminal ileum. No adequate test of the valve competencey can possibly be made in the afternoon unless the patient has fasted all day. I have administered the opaque injection to several thousand patients in accordance with all the precautions just enumerated, and I feel that the results obtained, especially with reference to the terminal ileum and the ileocolic valve, are satisfactory and comparable, because having been done under standard conditions and unvarying technic all cases can be compared with each other.

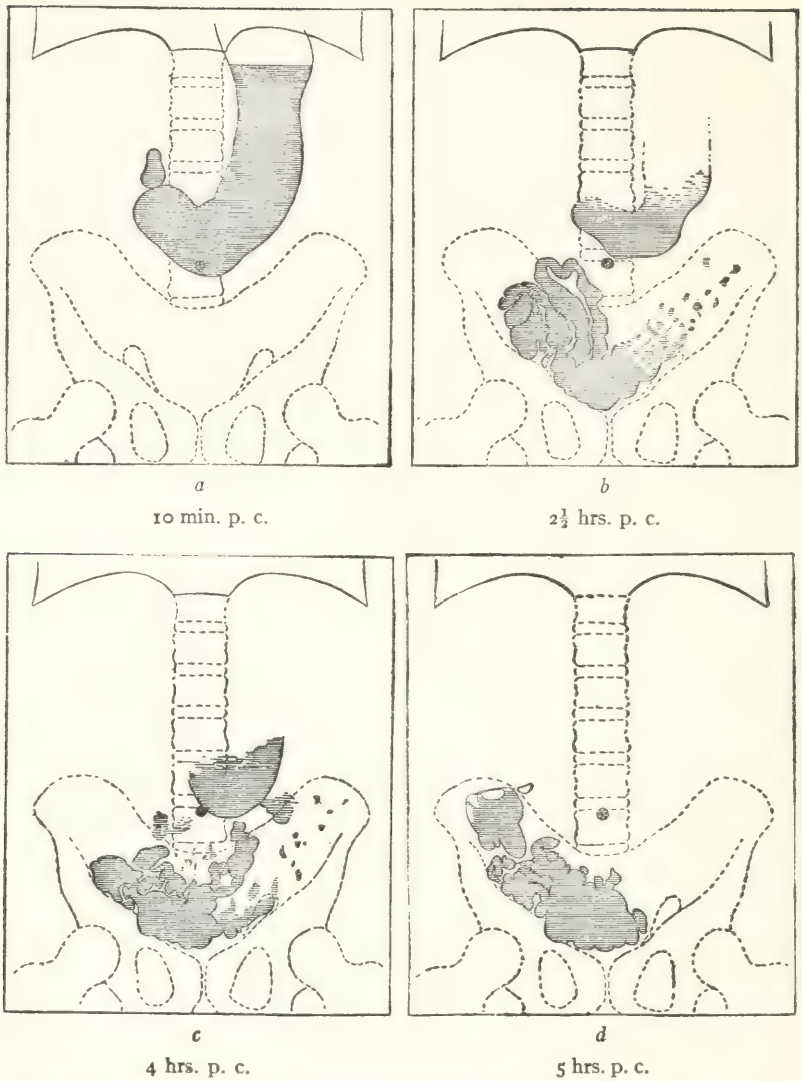
We will now proceed to the consideration of the various roentgenograms and tracings Dr. Moir has made for us in the case of the young woman whose anatomy we have been studying, and see if we can construct a "time table" relative to the passage of the opaque substance along the gastro-intestinal tube. I have grouped all these diagrams for convenience of comparison (Figs. 120 and 121, diagrams *a* to *h*, pages 824, 825).

Diagram *a* shows the filled stomach, with patient standing upright, about *ten minutes* after the ingestion of the opaque breakfast, as above described. This subject was an unusually well-developed young woman, and we see the lower border of the stomach about an inch below the navel with the patient in the

erect position. This is an unusual observation and, as I have previously declared in these lectures, if a stomach must be at this high level in the abdomen to be normal we encounter very few normal stomachs—at least so far as position is concerned. In one series of studies on 30 women students who believed themselves in normal health, I found the lower border of the stomach located in the average 2½ inches below the umbilicus, the patients being in the upright posture. In only 3 out of the 30 did the lower border of the stomach reach a point as high as 1 inch below the navel, and in these 3 there were other conditions that accounted for the unusually high position.

Diagram *b*, made *two and one-half hours* after the ingestion of the meal, shows about half of the meal in the stomach and the remainder in the small intestine, most of it in the terminal ileum, occupying the right and median thirds of the true pelvis.

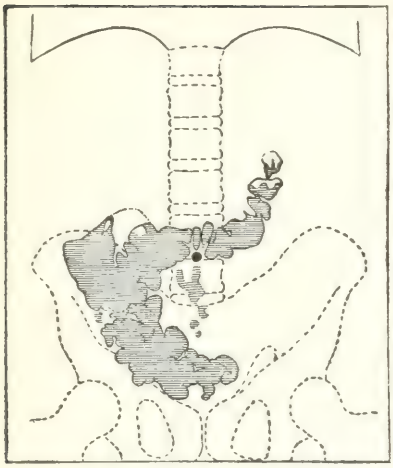
Diagram *c*, made *four hours p. c.*, shows a residue of about one-fifth in the stomach and still an appreciable accumulation in the terminal ileum. So far none of the barium has passed into the cecum. This illustrates very well the normal delay of food residues in the terminal ileum, and indicates that one should be slow in making a diagnosis of intestinal obstruction in segments of the intestine where these normal delays are noted. Thus, there is a certain delay amounting to a few seconds at the cardiac orifice. The pylorus normally offers a certain hindrance to the passage of material out of the stomach, so that four to five and one-half hours are required for gastric clearance. There is also a comparative delay in the terminal ileum, where Keith and others have shown a mechanism to exist for the regulation and retardation of the passage of material from the ileum into the colon. It has been shown by Cannon and others in animals, and by my own experiments and those of Boehm in man, that there exists in the transverse colon somewhat to the right of the midline a point proximal to which food is delayed for a considerable time. Arthur F. Hertz and others have shown that there is a normal delay of food residue in the pelvic colon, the latter forming a reservoir for the collection of fecal masses prior to their entry into the rectum just before and during the process of evacuation of the lower bowel.



a 10 min. p. c. *b* 2½ hrs. p. c. *c* 4 hrs. p. c. *d* 5 hrs. p. c.

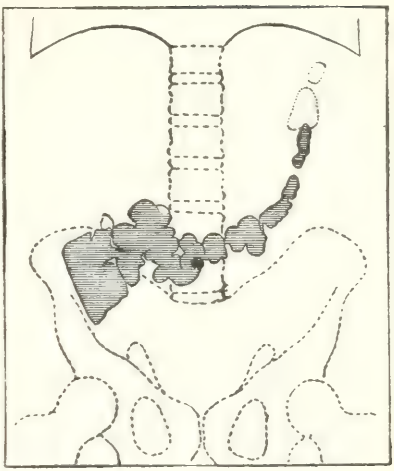
Fig. 120.—Tracings made at intervals following the ingestion of a barium meal (normal case).

Let us, therefore, recognize that a certain retardation of material in the terminal ileum just proximal to the ileocolic junction is to be expected under normal conditions.



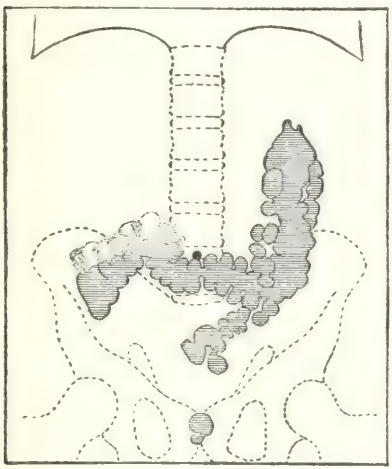
e

6 hrs. p. c. Bowels opened at 5½ hrs.



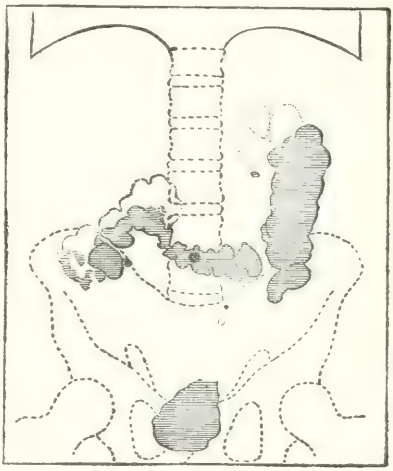
f

9 hrs. p. c.



g

26 hrs. p. c. Bowels opened at 25 hrs.



h

35 hrs. p. c. Incomplete b. m. at 35 hrs., just preceding observation.

Fig. 121.—Tracings made at intervals after the ingestion of a barium meal (normal case).

Diagram *d* has been made at the *fifth hour* of observation and shows the stomach quite empty. Most of the barium is seen in

the terminal ileum, occupying the right half of the true pelvis. A portion of the barium has passed through the ileocolic valve, occupying the cecum and ascending colon.

Diagram *e* (Fig. 121), made at the *sixth hour* of observation, shows the head of the barium column well along in the transverse colon, while there is still a definite residue in the terminal ileum. The proximal colon, comprising the cecum, the ascending and the right half of the transverse colon, shows rather marked distention as compared with the distal half of the transverse colon. This will be readily understood in the light of our previous demonstrations concerning the phenomenon of colonic antiperistalsis which tends to retard the passage of the colonic contents beyond a point somewhat to the right of the midline in the transverse colon.

Diagram *f* has been made at the *ninth hour*. It shows the terminal ileum empty, while the food residues are seen distributed throughout the transverse colon, reaching well up to the splenic flexure.

Diagram *g*, which represents the condition at the *twenty-sixth hour*, shows a rather uniform distribution of barium throughout the colon, with the exception of the lower part of the pelvic colon and the cecum which have been nearly emptied by a recent bowel movement, only a trace remaining in the lower part of the rectal ampulla.

Diagram *h*, made at the *thirty-fifth hour*, shows the disposition of the bariumized residues immediately following another attempt to evacuate the lower bowel. It so happened that the patient did not permit herself to complete the movement for fear of being late at the appointment in the roentgen examining room.

As I described in a lecture last spring on the "Roentgenologic Aspects of Intestinal Stasis" (Medical Clinics of Chicago, Vol. I, No. 5, p. 829), the principal propulsive movement in the colon is the "mass movement," first observed by Holzknecht in 1911, by which large masses having the size and shape of a sausage are carried at a rapid rate along the colon for considerable distances. I have demonstrated that as a part of their function these mass movements are set up either during or following the evacuation of the rectum in the act of defecation, and for this reason patients

should be urged to devote more time to the performance of the act. They should be instructed not to be satisfied with the first expulsive act, which may perhaps quite clear the rectum and ampulla, but to wait for the mass movements to bring down into the lower pelvic colon and rectum more fecal matter for expulsion.

Fig. 122.—Roentgenogram made twenty-six hours after a barium meal showing the filling defect in the colonic shadow immediately after the progression of a large segment of colonic content during a "mass movement." This irregular disposition of the barium is soon corrected, and within a quarter of an hour the residues are redistributed in a more or less regular fashion.

It requires a few minutes for this redisposition of colonic contents to occur, but with the necessary patience, and without the violent straining which some individuals seem to feel a necessary part of a complete bowel evacuation, more material will be brought down into the rectum, whence it will be evacuated without difficulty. A third or even fourth discharge may occur with the exercise of a

little more patience and perseverance. The time thus spent is not wasted by any means; on the contrary, it could not be more usefully employed. I have also observed that a little kneading in the cecal region aids in the stimulation of these important mass movements.

The irregularity of the distribution of the barium residues in the colon for a short time after one of these mass movements may cause confusion, and even simulate the defect in the colonic shadow caused by a malignant tumor (Fig. 122).

At the *fiftieth hour*, as we have seen, the colon has been completely emptied of opaque residues. I believe that the gastro-intestinal motility of this patient is normal. She would have completed the evacuation of the colon at the thirty-fifth hour under ordinary circumstances.

Briefly summarizing, the head of the barium column in a normal individual should reach the terminal ileum within two or two and one-half hours, pass into the cecum before the fifth hour, reach the splenic flexure before the ninth hour, and the rectum a little later, the exact time varying with the patient's bowel evacuations. Barium may be evacuated at the ninth hour or not until the following morning, just before observation at the twenty-sixth hour, depending upon the thoroughness of the previous evening's evacuation at or near the ninth hour. The last of the barium should usually leave the stomach before the fifth or certainly before the sixth hour, and all of it should be out of the ileum and in the colon before the ninth hour. The colon should be free from barium residues at the fiftieth hour.

The following is Groedel's table relating to the passage of the opaque meal through the gastro-intestinal tract, giving the average time of the appearance of the shadow at the various points of the digestive canal:

	Visible.	Empty or passed beyond.
Duodenum	5 minutes.	2 to 4 hours.
Jejunum	15 minutes.	3 to 5 hours.
Ileum	30 minutes.	4 to 6 hours.
Cecum	2 to 3 hours.	6 to 24 hours.
Right flexure	4 to 6 hours.	
Left flexure	4 to 12 hours.	
Rectal ampulla, at the latest	24 hours.	

These are average figures, of course, and are subject to variations either way. It is understood that a normal period of gastric evacuation is a fundamental condition for their validity. Groedel's figures would indicate a considerably more rapid passage than the "time table" which we have constructed for the young woman who so kindly lent herself to our demonstration, but in a large experience, covering several thousand cases, not all pathologic, I have come to feel that Groedel's estimate is a little short of the actual figures. Intestinal motility even more than gastric motility is dependent upon a large variety of factors. The time of evacuation is especially variable, and, as we have already seen in our class work, the behavior of the various sections of the colon depends largely upon the frequency and thoroughness of bowel evacuation. At times only the terminal colon and lower sigmoid are emptied by the defecatory act, while at other times practically the entire colon may be cleared. Usually, however, one defecatory act clears the intestinal contents beyond the splenic flexure; again, in other instances the greater part of the transverse colon will be emptied also. If only part of the opaque colonic residues is evacuated, the remaining part of the barium meal will progress rapidly and within a couple of hours be redistributed throughout the colon.

Observations on the motor phenomena of the small and large bowel are of the greatest practical importance, but, as has already been emphasized, these observations must be carried out upon the functioning intestine. Studies as to the size, shape, and position of the intestine carried out by means of the opaque enema are of certain value, especially in the study of intra- and extra-intestinal tumors and in the location of gross obstruction. The opaque enema also gives information regarding the competency of the ileocecal valve. In spite of my observations, which I consider very convincing, and in spite of the fact that similar observations have been recorded by Lewis G. Cole, Schwartz, Groedel, Dietlen, and others, certain eminent clinicians have expressed doubts on the subject. A discussion of the relative importance of ileocecal valve incompetency at this hour, however, must be dispensed with on account of the limited time at my disposal.

I have just said that the study as to the size, shape, and position of the intestine visualized by the opaque enema is of only limited value. The reason is that the enema causes a totally abnormal filling, and in nervous individuals it may produce exaggerated intestinal tonicity to such a degree as to simulate spastic conditions or even organic constrictions. In fact, the opaque enema influences the colon in almost every respect in an entirely different manner than the opaque meal. With the former, haustral markings are nearly always absent; with the latter, the haustral segmentation is always present except during the short period required for the execution of the spontaneous mass movements which constitute the principal propulsive activities of the colon. The position of the enema-filled colon is often quite abnormal, especially when the rectal injection has been made with the patient in the knee-chest position. The transverse colon particularly may be shortened and raised, presenting an appearance very different from that in the case of the functioning bowel. The knee-chest position for enemas is quite unnecessary, for unless there is some gross obstruction the colon fills easily when the patient lies supine.

Nevertheless, as a matter of routine and in order to be thorough in the work we are attempting, I make it a rule to examine the colon both by opaque enema and opaque meal in every case. When the findings following the barium meal seem to point to obstruction in the colon at a certain point, testing the colon by opaque enema will give additional evidence of considerable value and serve as a reliable check upon the barium meal findings. Observations of the colon immediately after the expulsion of the opaque enema will still further control records already made. These findings, especially when checked by all three methods, have seldom proved erroneous when the cases came to the operating table or autopsy.

The roentgen method when carried out in the thorough way just outlined will, in the hands of experts, give findings which are nearly always correct. If an organic lesion obstructs, or if it is sufficiently large to produce symptoms, it ought not to be over-

looked, and in several instances we have succeeded in locating a tumor of the colon before it gave rise to symptoms.

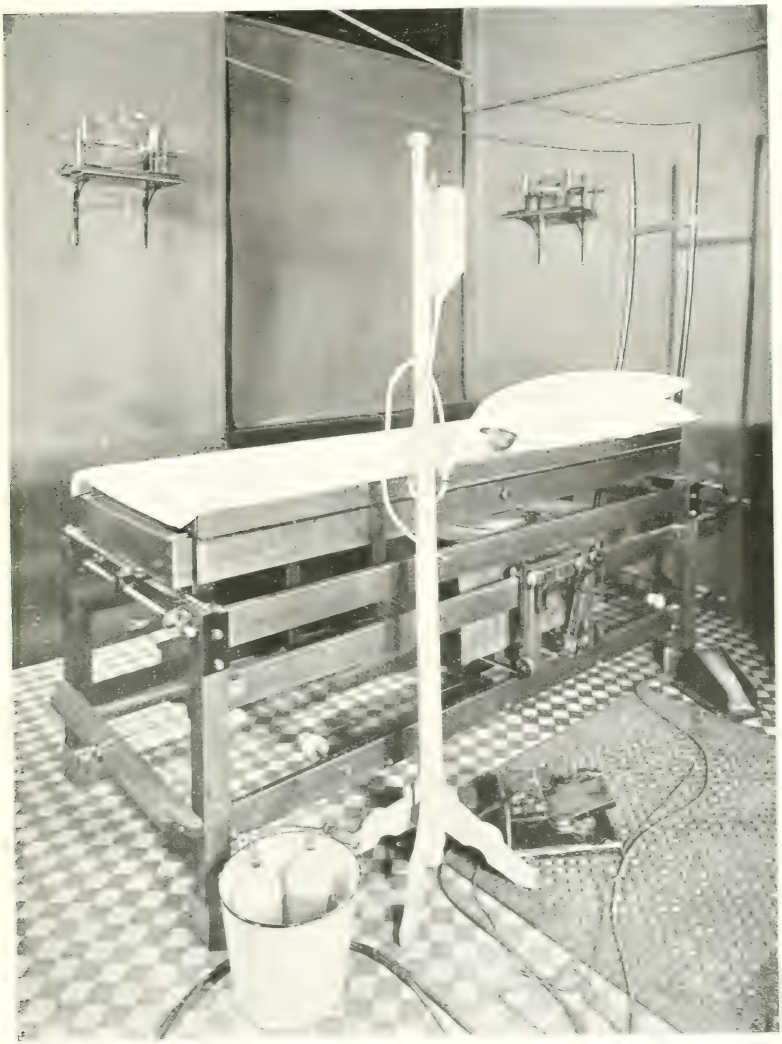


Fig. 123.—A satisfactory arrangement of horizontal fluoroscope and enema outfit for the administration of the barium enema.

The technic observed during the injection of the opaque enema is as follows:

It is important that the various requirements as described in an earlier part of this lecture shall be complied with. An ordinary enema can, with a capacity of 2 quarts and with 6 or 8 feet of tubing attached, is suspended at a height of 2 to 2½ feet above the table (Fig. 123). At the middle of this tube there should be an ordinary bulb syringe, with clamps upon the rubber tubing above and below the syringe. This syringe will be needed from time to time to clear out the tube of sedimented barium and to supply additional force should it be required. The enema point is an ordinary glass tube about 3 inches in length, supplied by pharmacists for this purpose. A supply of sterilized glass tubes may be kept on hand, so there will be no delay in changing tubes should a number of patients require examination in quick succession.

The opaque enema is prepared in 32-ounce bottles. The formula which I adopted some years ago, and which has been used in several thousand cases, is as follows:

To 2½ drams of gum tragacanth add about 1 ounce of alcohol. Shake well. Add 20 ounces of warm water and shake. Add 3 ounces of barium sulphate, then 20 ounces of water, shaking well each time. This mixture should be made up fresh shortly before using, but it will keep for several days in a cool place.

One 32-ounce bottle is usually enough to fill the colon, but in order to be prepared for emergencies it is best to have two bottles of the opaque suspension ready for use. Of course, the enema should be injected warm, preferably of a temperature of about 100° F. This temperature may be secured by having a bucket partly filled with warm water, in which the bottles may be warmed, and kept warm, until required.

Some roentgenologists prefer Haenisch's formula for the barium enema. This enema consists of water 1 liter, bolus alba 300 grams, barium sulphate 75 grams, and water sufficient to make 1 liter.

Holzknecht and Singer give the following formulæ: (*a*) Barium sulphate clysma: To 1 liter of boiling water a suspension of 2 soupspoonfuls of finest potato starch in ¾ liter of cold water is added, and, after being boiled again, 160 grams of barium sul-

phate and ¼ liter of hot water are stirred with it. The mixture is then boiled for five minutes and cooled off to 112° F. This mixture can be preserved in the ice-box for several days. (*b*) The bismuth clysma: To 1 liter of boiling water a suspension of 2 tablespoonfuls of finest potato starch in ¼ liter of cold water is added. This is boiled again for five minutes, and 120 grams of bismuth carbonate stirred in ¾ liter of cold water is added to it without boiling again.

Ordinary buttermilk, warmed to the temperature of 100° F., serves very well as a vehicle for the suspension of the barium for enema purposes. This latter preparation has the advantage of being quickly made ready.

In administering the barium enema the patient is placed supine upon a horizontal fluoroscope, so that the ray comes from below, illuminating a screen which lies upon the patient's abdomen or is suspended above it. In this way the progress of the injection can be watched as the fluid enters. Preliminary cleansing enemas are a requisite for these cases.

The patient having been prepared upon the horizontal fluoroscope, the rectal point is inserted not further than two or three fingerbreadths into the rectum. In fact, the ordinary rectal point which passes into the bowel only a couple of inches is quite satisfactory. When a tube is pushed up too high it folds upon itself and sometimes kinks and prevents the entry of the enema. Furthermore, it is unnecessary to use any form of colon-tube, as there should be no difficulty in filling the entire colon except in cases of organic obstruction.

As in all fluorsocopic work, no observations should be attempted until the eyes have been in the dark for a sufficient length of time, so that the enema tube in the rectum can be readily seen. The eyes having been prepared, the room is darkened and the current turned on. The stop-cock is then released, and in normal cases there appears in the pelvis a pear-shaped shadow which gradually increases in size, showing the filling of the ampulla. The distention of the ampulla having been accomplished, the opaque fluid begins to seek an outlet into the pelvic colon. Whether or not we shall be able to see distinctly the pelvi-

rectal junction and the last inch or two of the pelvic colon depends upon the location and disposition of this portion of the large bowel; according to its variable position the outlet from the ampulla appears sometimes toward the right, sometimes in the middle, and sometimes toward the left. When it appears toward the left it is usually possible to distinguish the pelvirectal angle very well, especially if the patient be turned slightly so that oblique illumination can be employed. When it appears toward the right or in the midline, the shadow of the opaque mass in the ampulla obscures the vision. In certain cases studies by means of the stereo plate method are exceedingly helpful.

It should be remembered that in almost every case, when the enema is injected with the patient in the supine position, there will appear to be a narrowing of the bowel where the pelvic loop passes over the iliopectineal line at its junction with the iliac colon. The appearance of narrowing is often exaggerated by the very short mesocolon possessed by this segment of the colon, and especially by the adhesions which often exist at this point. There first appear in the van of the advancing column several relatively small boluses, showing the regular spacings due to the indentation of the *haustra coli*. Under the increasing pressure, however, these haustral markings are smoothed out and the bowel shadow becomes broader, darker, and homogeneous. The normal pelvic colon rises higher in the abdomen during the injection of the enema, owing to the increasing pressure. When the rise of the pelvic colon fails to occur, it may be taken as evidence strongly suggestive of adhesions binding it down.

After entering the pelvic colon, and particularly after passing the iliopelvic junction into the iliac colon, the progress of the opaque stream is uninterrupted. The enema usually flows quickly up the iliac and descending colon, where it follows a course backward over the iliac crest, upward and forward around the splenic flexure, forward and downward across the transverse colon to the hepatic flexure. There is normally the appearance of retardation of the opaque stream during its passage around the splenic flexure. This is only apparent, as will be appreciated very easily if the patient be examined in the supine position with a tube

placed laterally, the screen being held vertically against the left side. Or, if the colon be studied stereoscopically, the two legs of the splenic loop (the last portion of the transverse and the first portion of the descending colon) are seen to be widely separated, the descending colon lying far dorsally to the last third of the transverse colon. The kinking which is supposed to occur often in the splenic flexure is rarely very sharp, but in those cases where it does occur the progress of the enema may be hastened by turning the patient upon the right side for a few moments. In the great majority of cases, however, it will not be necessary to turn the patient from the supine position in which the injection is started.

In the transverse colon it is sometimes observed that in the midline in front of the spine there is an apparent filling defect due to the saddling of the colon over the spinal column. This is especially noted in cases of increased intra-abdominal pressure.

In the hepatic region the fluid sometimes is observed to follow a very devious course, indicating a redundancy of the bowel at this point. Very rarely indeed is there any real kinking even when adhesions are numerous. Occasionally the fluid passes directly into the ascending colon, especially in well-nourished individuals; often it flows first upward in a direction parallel with the ascending and to its outer side, but later it returns and enters the ascending. Now the opaque fluid spreads rapidly, filling the ascending colon and the cecum, usually without difficulty. When the ileocolic valve is competent, the enema stops at the cecum, but in the rather large percentage of cases in which the valve is found insufficient the terminal coils of ileum are seen to fill for a greater or less distance.

In brief résumé, it may be stated that the normal colon can be filled by injection within a few minutes, the patient reclining upon the back, without elevation of the pelvis or the use of the colon-tube. There are certain physiologic obstacles which can be overcome by waiting, by manipulation, and sometimes by change of position. The greatest expansion is noted in the ampulla and sometimes in the cecum. Excepting the ampulla, the caliber of the colon, thus filled by opaque enema, gradually increases from

3 cm. in the pelvic colon to 6 cm. in the cecum. The larger the amount of the injection, the more redundant the colon, for it expands not only in breadth but also in length.

Normally, the opaque enema, which is perfectly bland and unirritating, proceeds through the entire colon to the cecum without exciting any visible peristaltic contractions, but in the conditions

Fig. 124.—Pseudofilling defect in ascending colon (enema filled) due to peristaltic contraction.

of inflammation or overdistention characteristic ring contractions may be excited. When seen, these have diagnostic value and sometimes may cause confusing pseudofilling defects (Figs. 124 and 125). Normally, several minutes elapse without such peristaltic waves, even after the head of the enema column has reached the cecum. The haustral markings, which have been smoothed out during the entry of the enema, usually return after some time,

occasionally in an instant, but usually more slowly, beginning first in the distal colon.

Following the study of the enema-filled colon it is often very instructive to watch the emptying of the enema or to study the colon immediately after the expulsion of the enema (Figs. 126 and 127). Haenisch advises that the container from which the injection has been made be lowered, and the enema allowed to

Fig. 125.—Pseudofilling defect in ascending colon (enema filled) due to peristaltic contraction.

return by gravity. The emptying of the colon is thus watched under the fluorescent screen. This is often very inconvenient, however, and most of the aid thus obtained can be secured by a screen study immediately after the patient has expelled the enema.

Let us briefly summarize the different facts concerning the stomach and bowel which may be learned at each of the obser-

vations following the ingestion of the barium meal. The observations on the thorax, esophagus, stomach, and duodenum have already been carefully described in a preceding lecture, so no more time will be devoted to those points here.

At *two and one-half hours* we will be able to estimate the rate of gastric clearance and the appearance of the upper small bowel, at the same time paying attention to whether or not the head of

Fig. 126.—Roentgenogram of normal colon, patient prone, immediately after the introduction of the opaque enema.

the barium column has reached the ileocecal region. If any question with reference to the duodenal bulb should still be unsettled in the observer's mind, new observations are made in regard to defective bulbar filling and mobility of the duodenum, especially of the first portion.

At the *fourth hour* the stomach is usually found empty or nearly so, and the patient is permitted to take food. If the

stomach is not yet empty, an estimate is made of the time which will probably be required to completely clear the stomach, and the patient is requested to refrain from taking food until after the expiration of the estimated time. If necessary, another observation may be made at the sixth hour to gauge more accurately the exact emptying time of the stomach. Practically,

Fig. 127.—Roentgenogram of the same colon shown in preceding figure after the patient's successful effort to expel the enema. The outline of the colon may be made out, as a trace of the barium still is attached to the walls. This colon has been practically cleared by the expulsive effort, as should be the case in normal individuals.

however, this may be just as accurately observed when the patient returns for the ninth-hour study, for I have discovered by long experience that whatever portion of the barium meal remains in the stomach at the time the patient eats lunch will still be there at the ninth-hour observation. For instance, if the patient has a two-hour residue in the stomach at 2 o'clock when he has permis-

sion to take lunch, that barium residue will remain in the stomach until the lunch has been evacuated, no more barium passing out of the stomach until near the end of digestion of the lunch.

At this time, the fourth hour, one further notes the appearance and movability of the various coils of jejunum and ileum, and pays special attention to the character of the segmentation movements going on in the terminal ileum. Special manipulation is required for testing the mobility of the last 8 or 10 inches of ileum, that portion which rises out of the massed coils of ileum occupying the true pelvis, and reaches up over the iliopectineal line to join the cecum. This terminal loop of ileum has been called the "long arm of the ileum." By carefully manipulating the abdomen under fluorescent screen guidance one may determine the presence or absence of binding adhesions of the terminal ileum, whether or not the cecal and terminal ileum shadows may be separated, whether the appendix shadow shows, and, if visible, whether or not it is fixed. Points of tenderness on pressure may be identified in relation to the visualized bowel. The cecum may be studied as to contour, size, position, mobility, and coincidence with points of tenderness on pressure.

At the *ninth hour* one may begin to form an opinion as to the degree of antiperistaltic activity in the colon; iliac stasis, as shown by the presence of a residue in the terminal ileum which should have been cleared by this time; the size, contour, and mobility of the cecum, and possibly also of the appendix; the mobility and caliber of the ascending colon, and possibly the mobility of the transverse colon. It is usually possible at this time to test the separability of the two legs of the hepatic flexure; these should normally be easily pushed apart with the manipulating hand, and when this cannot be done it is logical to infer the presence of binding adhesions. A note should be made as to the number and character of bowel movements since the opaque meal was eaten.

The *twenty-sixth-hour* observation gives very important information. Again the patient will be questioned relative to the number and character of the bowel movements since the beginning of the barium meal, as the appearance of the barium residue

in the bowel must be interpreted accordingly. Special points of hindrance will be noted. A lookout will be maintained for the spontaneous mass movements already referred to, as well as for antiperistaltic manifestations. The cecum and the appendix will again be studied, and a new observation made with reference to cecal and appendical mobility. The terminal ileum will also be carefully studied for possible opaque shadows, the presence of which will be pathognomonic of marked incompetency of the ileocecal valve. A new note will be made concerning points of pain on pressure. Special attention will be devoted to the iliac and pelvic colon, for in the majority of instances it is probable that the cause of constipation, if mechanical, will be located below the crest of the left ilium, involving either the iliac or pelvic colon or the rectum itself. Rectal constipation is a very common finding.

At the *thirty-fifth* and *fiftieth hours* similar observations will be made, special care being exercised to quiz the patient with reference to the number, time, and character of bowel movements, for such information is highly essential to a proper interpretation of the roentgen findings. The colon should have been emptied before the fiftieth hour. If such is not the case, the disposition of the residues should be carefully noted, especially whether the larger portion is in the proximal colon, such as will happen in cases of exaggerated antiperistalsis, or whether the greater mass is in the distal colon, perhaps even in the rectal ampulla itself. At times cases are found in which both types of stasis are combined.

Although adhesions of the pelvic colon are probably the most disturbing of all adhesions likely to be found in the abdomen in relation to the alimentary tract, they are the most difficult to recognize, at least by the ordinary process of manipulation with the protected hand under screen guidance. In the majority of cases, therefore, other methods of determining the presence of disturbing adhesions of the pelvic loop must be depended upon. This is possible by resorting to manipulation as much as the case will permit, and by comparing the height to which the pelvic loop rises during the injection of the opaque enema. This is the prin-

cipal reason for insisting that the injection of the barium enema be always done under screen guidance. The fixed pelvic loop will not rise in response to the pressure necessary to fill the entire colon with enema, whereas the top of the normally movable pelvic loop will rise several inches higher during the injection. Following the enema, the patient will usually have difficulty in evacuating the enema except below the point of binding adhesions. Following the meal there will usually be colonic delay, for the most part in the proximal colon, not in the distal large bowel; and if the delay occurs in the distal bowel it will be evident above the point of hindering adhesions.

Once again let me emphasize the necessity of collating the other laboratory and clinical findings with the roentgenologic observations. The mere determination of intestinal delay does not mean that the patient is the victim of alimentary toxemia. The roentgen method simply adds another—very powerful and valuable—means to the other resources of diagnostic medicine, and we should be careful not to expect too much of the roentgenologist, nor to depend upon his findings to the exclusion of other tests which experience has shown valuable in individual cases.

QUESTION: How many roentgenograms do you make or have made in the average examination of the gastro-intestinal tract?

DR. CASE: In some cases none; in other cases six to ten, depending upon the interest and importance of the case and the size of the patient. In very heavy patients I am not willing to jeopardize my judgment of the duodenal bulb for the sake of a few roentgenograms. I always depend upon plate studies for an opinion as to the presence or absence of gall-stone shadows. Occasionally I feel it necessary to make a few plates for the remainder of the examination, but in the majority of instances studied in the hospital my conclusions are based entirely upon screen observations, made for the most part with the patient in the horizontal position. In the days when we attached more importance to the position of the transverse colon and the lower border of the stomach, examination in the upright position was, of course, essential, but since we have come to recognize that it is of far greater importance to study the function of the bowel than its position, the

necessity for studies in the erect position is no longer apparent. The horizontal fluoroscopic table permits of a very much more satisfactory examination of the patient. In the erect position the abdominal muscles are more or less tense in spite of any effort of the patient to relax, and satisfactory manipulation under the screen in this position is almost impossible.

QUESTION: You do not seem to approve of the high enema. Will you give us your idea of the effectiveness of an enema and how it should be administered?

DR. CASE: In other lectures of this course we have learned that colonic stasis occurs preferably in two places, viz., in the pelvic colon and in the cecum. Cecal stasis is common in conditions of chronic appendical disturbance with or without adhesions, in those rare cases of Jackson's membrane where the upper fibers constrict, and particularly in that large class of individuals where, through spasticity of the pelvic colon, there is secondary distention and delay in the right half of the colon. That these are the usual sites of stagnation is evidenced by the fact that carcinoma occurs most frequently in these two portions of the bowel. Carcinoma rarely occurs in the transverse colon, which would naturally be the seat of stasis should the delay be found due to the drag of the ptosed colon upon the high flexures. The remedy for stasis in the proximal half of the bowel, excluding, of course, those instances of appendical involvement with pericecal adhesions and obstructing bands along the ascending colon, is a therapy to overcome the spasticity of the iliac and pelvic colon.

A different therapy is needed in cases of rectal and pelvic colon stasis. Stasis in the rectal ampulla, excluding local lesions, occurs only through neglect to respond to natural reflexes. The resulting condition of rectal stasis is termed "dyschezia." It seems unreasonable to administer a laxative which first irritates 25 feet or more of bowel in order to evacuate the contents of the last 12 or 15 inches of the colon. Here enemas are indicated, and small enemas at that. A small, cold enema is often enough to produce a thorough evacuation of the bowel in these cases. Such patients need training or educating, special emphasis being laid upon the necessity of responding to the slightest stimulation of the defeca-

tion reflex. If the cause of the rectal stasis is an irritating or inflammatory lesion, a warm enema should naturally be given the preference.

The "high" enema is a misnomer, and it should rarely, if ever, be attempted. The work of H. Wellington Yates, of

Fig. 128.—Roentgenogram of rectal tube coiled up in rectal ampulla. The bowel also contains some barium residues, the largest shadow being caused by terminal ileum. It is impossible to pass a colon-tube into the iliac or descending colon without the aid of the surgeon's hand within the open abdomen.

Detroit, which has been confirmed by numerous investigators, shows that it is impossible to introduce a colon-tube higher than the junction of the iliac and pelvic colon (Fig. 128). Even with sterile hands working within the abdomen it is very difficult for the surgeon to pass a tube, put into the rectum by an assisting nurse, into the descending colon.

Furthermore, the colon-tube is quite unnecessary for a high enema. Perhaps 7000 times have we watched the introduction of the barium enema and found that only in cases of organic bowel obstruction did the head of the barium enema fail to reach the cecum within a few moments. The average amount of fluid necessary to reach the cecum was 1200 c.c., the necessary pressure being supplied by the enema container being raised 2½ feet above the patient. No colon-tube was ever employed—only an ordinary rectal point, inserted just past the sphincters, the patient lying flat upon the back. Very rarely indeed has it been necessary to resort to the knee-chest position. When the knee-chest position is used the resulting distortion of the colon may be very confusing. In the light of our experience it is difficult to understand how harm can fail to result from the introduction of such large enemas as one often hears of. A 2-quart enema is a very large one; a 3-quart enema exceeds the limits of safety.

QUESTION: Why bother with the enema when one can easily empty the bowel by means of laxatives?

DR. CASE: Practically all laxatives, which are irritant in their action, are seen to increase the spasticity which is so common in cases of constipation. I remember one very interesting case which will illustrate my meaning. A patient was referred to me for suspected carcinoma not involving the pylorus. The examination, which resulted in a positive diagnosis, included a study of the bowel according to our custom. The head of the barium column reached the hepatic flexure by the fourth hour and the splenic flexure by the ninth hour. By the twenty-sixth hour the right half of the bowel was already partially emptied, and by the fiftieth hour the colon had been completely cleared of the barium residues. In other words, the behavior of the colon was quite normal. The colon was normal in size, shape, and position. The pelvic colon was freely movable, and spasticity was not mentioned among the findings. This examination was, in accordance with our custom, carried out without the preliminary administration of laxatives. Several days later permission was given to operate. In order to secure a stereoroentgenographic record of the tumor I called for the patient the day before the operation

and gave him a new barium meal. Having made the stereoroentgenograms, the patient was given the usual dose of magnesium citrate in order to clear out the bowels in preparation for the operation. Instead of emptying easily, as was the case during the previous examination, there was now observed a notable distention of the cecum, ascending colon, and right half of the transverse colon. Antiperistaltic waves were seen in the right half of the transverse and in the ascending colon. The left half of the colon was very spastic, whereas spasticity had not been mentioned among the previous findings. The spasticity in the pelvic colon, the antiperistalsis, and the resultant delay in the cecum and ascending colon were so marked that even after the administration of fifteen or sixteen enemas the patient went to the operating room without having cleared the cecum and ascending colon of the barium residues. At the operating table I was able to demonstrate an enormous distention of the cecum, ascending colon, and right half of the transverse colon, as well as the presence of considerable barium-mixed fecal matter.

This is only one of many similar observations, our findings confirming the clinical studies of Abt, who found that all the common laxatives were irritants. Most constipated patients need antispasmodics, not irritants, and for this class of patients belladonna is more appropriate than cascara. Most useful of all preparations in these cases is paraffin oil in one of its various forms. My experience with paraffin oil has been very satisfactory. The use of agar-agar has also been attended by beneficial results. The plain agar-agar serves best, except in the relatively rare cases of atonic constipation. The addition of cascara to the agar-agar produces the same irritant effect as the ordinary laxatives. Briefly stated, I am convinced that the common drug laxatives are contra-indicated in most cases of constipation, for, contrary to the older ideas, our roentgen studies have shown that in the majority of cases bowel inactivity is due not to atony of the intestinal musculature, but to some conditions associated with hypertonicity; that if medicinal aids are required, some form of paraffin oil and the agar-agar preparations are more appropriate, perhaps with the addition of an antispasmodic, as belladonna.

QUESTION: What do you use in place of laxatives to clean out the bowels before the bismuth study?

DR. CASE: Unless the patient is very constipated we do not believe in the administration of laxatives or non-medical remedies for opening the bowels. A knowledge of the condition of the bowels before the administration of the roentgen meal is important, and the advisability of giving laxatives preceding the opaque meal is a much-debated point. Gompertz and Richard have found no appreciable difference in either case, but, inasmuch as their test was confined to a paltry 4 cases and directed to motility only, which is but a fractional part of the roentgen findings, their investigations are devoid of practical significance. No doubt more extensive research would have revealed very decided changes resulting from the use of laxatives.

As an argument in favor of laxatives A. F. Hertz states that we wish to see the patient under natural conditions, but that is precisely the reason why laxatives should not be used. Hertz correctly points out that most cases of obstipation are more or less cumulative, that the bowels are never completely evacuated, that more and more feces are retained, increasing from below upward. He correctly concludes that partial obstruction may occur in the higher parts of the colon as a result of such accumulation, although the motor functions of the intestines as far as the pelvirectal flexure may be normal, and that, consequently, the activity of all parts of the intestine can only be properly investigated after the colon has been emptied. But all these pathologic conditions are the very ones we want to study, while the condition of the empty colon, after the pathologic obstruction has been removed, is a matter of secondary interest. The pathologic fecal accumulation represents the natural conditions which may demonstrate exaggerated antiperistalsis, a dilated elongated cecum, an incompetent ileocecal valve, ileac stasis, etc., while removal of the accumulated feces would only demonstrate rectal constipation owing to the barium readily passing into the distal half of the colon, with the result that a very important factor in the diagnosis of the case is lost sight of.

CLINIC OF DR. ARTHUR F. BEIFELD

COOK COUNTY HOSPITAL

PURPURA HÆMORRHAGICA (WERLHOF'S DISEASE)

Differential Diagnosis of a Case Characterized Principally by Intense Bleeding from the Gums and by a Profound Anemia. Presence in the Blood of Pathologic Lymphocytes or Lymphoblasts. Correct Interpretation of the Blood-picture and the Clinical Phenomena. Fatal Outcome after Six Transfusions of Blood.

THE history in this case is brief and the physical findings relatively meager. This pallid young man is thirty-two years old, a native of Russia, married, and a laborer by occupation. During the past four months he has been working in a starch factory in a town not far from Chicago. He came to the hospital because of two manifestations, namely, bleeding from the gums and reddish spots on the skin.

The reddish spots—we may anticipate a little and call them hemorrhages—appeared about two months ago, first on the legs, and later became more or less generalized over the body. Three weeks ago his gums began to bleed and have continued to do so almost constantly since. A week before admission the patient noticed a swelling in the region of the right cheek bone, the prominence of which pointed inwardly toward the mouth. Thinking this was an abscess, he tried twice to open it with a needle, but obtained each time only a considerable amount of thick dark blood.

The patient has had hemorrhages from no other mucous membranes; and despite his very evident anemia he complained

at first of none of the symptoms usually accompanying the latter, and now is troubled only with dyspnea, headache, and insomnia. This may perhaps be referred, in part at least, to his limited vocabulary. In particular, there is nothing pointing to the existence of joint disturbance.

His previous history contains nothing of importance. There is no history of rheumatism. The family history is likewise negative. Venereal disease is denied. He has two healthy children and his wife has had no miscarriages. A moderate use of alcohol is admitted.

You will see from the temperature chart that the patient has had practically no fever since he came to the hospital. At irregular intervals the curve has risen slightly above the normal line, but has never reached 101° F. The pulse, however, has been consistently rapid and has only infrequently fallen below 100. At present it is rapid, 62 to the half-minute, small, compressible, regular, and of rather low tension. The blood-pressure readings are 110 systolic and 80 diastolic.

Physical Examination.—The patient is a fairly well-nourished individual, distinguished chiefly by his extreme pallor. The skin is of a dead white color and the visible mucous membranes are almost bloodless. On the arms and legs, in the axillary regions, and over parts of the chest and back are fine hemorrhages, while on the arms and back are a few areas we should call ecchymoses. The ankles pit slightly upon pressure.

The lymph-glandular system has been carefully examined from day to day, and has revealed nothing in addition to what was observed when the patient entered the hospital, namely, a few small, non-tender nodes in the upper cervical chains on both sides and a gland or two in the inguinal regions.

The positive findings about the head and neck are the following: A small subconjunctival hemorrhage on the nasal side of the right eye at the sclerocorneal junction; a small retinal hemorrhage on the temporal side of the left disk, with extremely pale nerve-heads on both sides; gums which are slightly swollen, not tender, at some points oozing blood, and at others covered with dried blood-clots; and a large submucous hemorrhage in the

upper jaw on the right side, extending from the canine tooth backward and upward, and manifested externally by a firm infiltration about the malar bone.

The chest shows no changes aside from the presence of many subcrepitant râles over the base of the left lung posteriorly. There is a soft, non-valvular murmur, systolic in time, heard best over the apex and pulmonic area. The abdomen is negative to palpation, with the exception of a slightly enlarged spleen. The area of splenic dulness, however, is distinctly increased, extending upward to the seventh rib, downward and forward to the anterior axillary line, and merging posteriorly with the lumbar dulness. There is dulness a fingerbreadth below the costal margin in the splenic zone, and on deep inspiration the edge of the organ can just be felt. More important than this is the fact that the spleen could not be palpated when the patient entered the hospital, nor was the area of splenic dulness appreciably enlarged.

The remainder of the physical examination is entirely negative. The urine and sputum contain nothing pathologic, the cerebrospinal fluid is normal in every particular, and the Wassermann reaction on the blood is negative. The leukocytes on the day of the patient's admission into the hospital numbered 3400.

The course of the disease during the patient's stay in the institution has been characterized chiefly by variations in the intensity of the bleeding from the gums. This variation has been dependent upon the therapy employed, and will be considered more in detail later in connection with the subject of treatment. In general, it may be said that the patient has shown a progressive decline, marked by an increasing asthenia, cardiac enfeeblement, inability to retain nourishment, and periods of stupor.

DR. BEIFELD (addressing Student): Mr. A., with this information before you, what diagnosis would you suggest? The patient was sent from the examining room, where there is only a brief opportunity for the study of each case and no laboratory facilities, with a tentative diagnosis of "purpura hæmorrhagica." What is your understanding of this condition?

Mr. A.: It is a disease associated with bleeding from the mucous membranes.

Dr. Beifeld: Yes, that is one of its chief characteristics. Incidentally, it is also known as *morbus maculosus*, or *Werlhof's disease*. It is said to occur most commonly in young adults, especially in girls, and to attack by preference weak, undernourished individuals, though those in previous good health are by no means immune. The course is variable. Some cases are rapidly fatal, others recover, while still others are reported to run a chronic course, lasting for months.

The immediate cause of purpura hæmorrhagica is not known. Recent studies have shown, however, that it has been made to include a number of totally unrelated conditions, chief among which are various septic processes and the acute leukemias. Though the future may show that the disease is merely a symptom-complex, for the present at least we must retain the nomenclature, applying it to what may be called typical examples of the condition, and carefully excluding diseases which improved methods have shown to belong elsewhere. One almost constant feature of such typical cases is a neutrophilic leukocytosis. In our patient, as you will recall, the white blood-corpuscles numbered 3400 per cubic millimeter when he entered the hospital, and, as we shall see, the count has constantly been even lower than that figure in later enumerations. Therefore, though our case, because of the age, the hemorrhagic diathesis, and especially the tendency to bleeding from a mucous surface, might be loosely cataloged as one of purpura hæmorrhagica, it is evident that it is not a *typical* example of this disease.

A word now as to sepsis, which we have mentioned as a possible cause of certain cases of morbus maculosus, and which, incidentally, may play a part in the causation of the acute leukemias, themselves not infrequently miscalled purpura hæmorrhagica. Recent years have so greatly enhanced the etiologic importance of septic diseases that it is becoming increasingly difficult to eliminate them as factors, whether primary or otherwise, in the individual case. The patient before us exhibits a number of the earmarks of an infection, namely, fever, splenic

tumor, and the hemorrhagic diathesis. But speaking against sepsis are the leukopenia, the apparent absence after careful search of a focus of infection, and repeatedly sterile blood-cultures, while the intensity of the gingival hemorrhages seems to indicate that some condition other than a septicemia, as we at present understand this term, is responsible for the patient's illness.

Assuming, then, that a diagnosis of purpura hæmorrhagica or of septicemia does not satisfy all of the conditions we have before us, what else in the way of diagnosis would you suggest?

MR. A.: I should think strongly of scurvy because of the condition of the gums.

DR. BEIFELD: Very true. Scurvy is another of the hemorrhagic diseases that must be considered. Scurvy, by the way, is a disease illustrating very well what I told you a moment ago concerning the growing importance of septic processes in the etiology of conditions hitherto considered as of non-infectious origin. A lack of certain essentials in the diet unquestionably plays a part in the production of scorbutus, yet this factor is not entirely satisfactory in explaining certain symptoms of the disease which harmonize better with the assumption of an infection.

Please examine the patient's gums and tell me whether they are characteristic of scurvy. Sponge the bleeding surface with this piece of gauze.

MR. A.: The gums are very pale. There are no definite bleeding-points, but only a general oozing. The crusts that are present seem to be only clotted blood, for the mucosa beneath them is smooth and glistening. It is my understanding that the gums in scurvy are red and swollen, especially between the teeth; that they bleed very readily when touched, and that there is often considerable ulceration present.

DR. BEIFELD: Your description is first class. In the early stages of the disease, however, the gums may show little except a hyperemia. The scorbutic changes are most marked in the projecting parts of the gums between the teeth; in fact, they are generally absent where teeth are missing and in old people and children who have no teeth. The ulceration may go on to

great destruction of tissue and may terminate in a foul ulcerative stomatitis.

Were this a case of scurvy we should expect, after a duration of nearly four months, to find the gums red, swollen, spongy, and ulcerated, a condition which is not present here. But granting that the gums are not seriously involved, as happens not infrequently in scurvy, making the diagnosis of the sporadic case very difficult, what other manifestations do you expect to find?

Mr. A.: Hemorrhages into the skin.

Dr. Beifeld: The superficial hemorrhages are not especially characteristic unless it be their tendency to localize about the openings of the hair-follicles. The deep hemorrhages, on the contrary, are almost pathognomonic of scurvy. Even in the absence of gum changes these deep infiltrations are practically diagnostic. Their site of election is in the loose tissues about the malleoli and the lower ends of the femurs, where they form board-like tumors, which may or may not extend into the overlying skin. In the latter case they exhibit the various color changes characteristic of disintegrating blood. Though these deeply situated masses are seen most frequently in the lower extremities, they may also occur in the arms and on the trunk.

The gum changes and the deep hemorrhages are the two most characteristic manifestations of scurvy and are present in the great majority of cases. Neither is present in our patient, and for this reason we need feel no hesitation in ruling out the possibility of scorbutus.

Before we go further let me give you the results of the successive hematologic examinations in this case:

Date.	Hemoglobin (Sahli).	Erythrocytes.	Leukocytes.
4/26/16	35	1,800,000	3400
5/3	15	600,000	2600
5/6	30	1,400,000	1600
5/8	36	1,720,000	1000
5/11	30	1,420,000	1400
5/13	40	2,100,000	2600
5/16	..	1,680,000	2000
5/23	25	960,000	1400
5/27	13	580,000	1000
5/30	10	440,000	1000
5/31	10	400,000	1200

In order to make the estimation of the hemoglobin as accurate as possible when the percentage was extremely low, two or more pipetfuls were used and the result divided by the number employed.

Three things stand out in this table: The high grade of anemia present, the high color-index—at times considerably above 1 and rarely falling below 1—and the marked leukopenia.

The distribution of the leukocytes on the same dates was as follows:

	4/20	5/3	5/11	5/20	5/27
Polynuclear neutrophils	38%	20%	32%	25%	32%
Lymphocytes	30	56	16	30	40
Large monos and transitionals	8	4	12	8	6
Eosinophils	0	0	0	1	1
Basophils	0	0	0	0	1
Myelocytes	0	0	0	0	0
Large mononuclear cells with homogeneous, basophilic cytoplasm	24	20	40	34	20

The erythrocytes varied considerably in size, there being many cells smaller than the normal and nearly as many of the macrocytic type. Poikilocytosis was marked in every specimen, and most of the slides showed some polychromasia and basophilic granulation. In these various examinations 4 nucleated red cells were found—3 normoblasts and 1 megaloblast.

The blood-platelets were noticeably increased in every specimen.

If I should ask you now for your diagnosis, I think you would not hesitate to say pernicious anemia. Speaking for this are the following: The tendency to hemorrhages, both cutaneous and from mucous surfaces; the profound anemia, red cell counts below 1,000,000 always suggesting the pernicious type; a color-index almost constantly greater than 1; the presence of many megalocytes; the finding of 1 megaloblast in a case in which nucleated red cells are extremely few, and the leukopenia with a relative prominence of mononuclear forms.

Why, then, is this not a case of pernicious anemia? In my opinion there are two very strong reasons speaking against this diagnosis: The first is the presence in our case of an abnormally

large number of blood-platelets, which, as I emphasized in an earlier clinic, are regularly diminished even to the vanishing point in pernicious anemia; and the second and weightier reason is the character of the cells which are classified as large mononuclear leukocytes with a homogeneous basophilic cytoplasm.

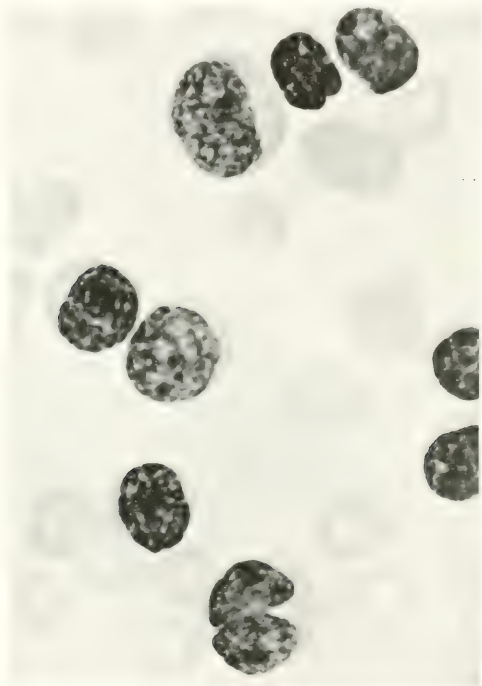


Fig. 129.—Composite picture illustrating the various types of lymphocytes found in this case (acute lymphatic leukemia). To the right are four practically normal lymphocytes. Above and to the left are three pathologic lymphocytes, the lymphocytic character of which is unmistakably shown by the coarse, knobby nuclear chromatin. Below is a pathologic lymphocyte with several deep nuclear clefts—the Rieder type. (Magnification × 1200.)

A stained smear from the blood of our patient is under the microscope. The field I have chosen is exceptional for this slide, in that it shows three of these cells in close proximity. In the remainder of the preparation one must search a long time because of the marked leukopenia to find more than one white cell in a

single field, using the oil immersion. These leukocytes (Fig. 129) have the following characteristics: In size they vary between that of a normal lymphocyte and that of an ordinary polymorphonuclear leukocyte; they resemble the lymphocyte in that the nucleus has a rather clean-cut outline, the nuclear chromatin is coarse and knobby, and the nucleus generally occupies the greater part of the cell body; and azure granules are common in the cytoplasm when the Giemsa stain is used. There is usually only one nucleolus seen, and many of the cells have instead of a round nucleus one with deep indentations (Rieder cells).

These cells are sometimes improperly called large lymphocytes. This term should be restricted, however, to normal lymphocytes, which are large because they are old. The cells we have been describing are pathologic lymphocytes or lymphoblasts, and represent the forerunners of the lymphocytes normally found in the blood.

Cells of this type may be found here and there in pernicious anemia, but never in the high percentage seen in this case. For this reason particularly I believe we are justified in eliminating pernicious anemia as a diagnostic possibility.

On the other hand, pathologic lymphocytes when present in large number, as here, are highly suggestive of another condition which we have already mentioned as being often mistakenly diagnosed as morbus maculosus. I refer to acute lymphatic leukemia. You may object to this diagnosis because of the marked leukopenia present. In "Medical Clinics of Chicago," Sept., 1916, p. 404, I have called your attention to the fact that leukemias, both acute and chronic, may run an *aleukemic* course, that is, the changes in the lymph-nodes, spleen, bone-marrow, liver, kidneys, and other organs are typically leukemic, with a normal or subnormal total white cell count in the circulating blood. In these aleukemic forms, however, the differential count is generally the decisive factor, for just as in the typical cases with a marked increase in the number of white cells per unit volume, so here there is present a very large relative number of lymphocytes. In the chronic lymphatic leukemias the cell type, which as a rule predominates, is the small one, while in the acute forms there are

seen, in addition to lymphocytes of normal or about normal size, many large cells.

This, as you will note, is the condition in our case. The combined lymphocyte count (normal plus pathologic cells) varies from 54 to 76 per cent. Furthermore, all of the remaining features of the blood to which we have called attention are fully consistent with the diagnosis of an acute leukemia. There may be a very profound anemia; the blood formation may assume the embryonic type just as in pernicious anemia, with a high color-index and many megalocytes. (This is frequently the case, though by no means always, for there may be little or no anemia, or the anemia may be distinctly of the secondary type.) Finally, nucleated red cells, both normoblasts and megaloblasts, poikilocytosis, polychromasia, and basophilic granulation may, one or all, be found in acute leukemia.

You are probably asking yourselves how a diagnosis of lymphatic leukemia is justified in the absence of more prominent lymph-node involvement. While this is not common, it does occur, and is by no means inconsistent with such a diagnosis, as a number of anatomically verified cases have shown. Acute lymphatic leukemias, both aleukemic and of the usual type, may run their course with little or no appreciable enlargement of the lymph-nodes or of the spleen. Cases with insignificant or no splenic and glandular involvement are known, furthermore, in which the blood has remained quite normal, both as regards the total number of white corpuscles in the cubic millimeter and their percental relations, until just before death, when the leukemic nature of the process became evident. Such cases, unless given the most intensive study, hematologically and histologically, are generally regarded as unquestioned examples of morbus maculosus, or, if the lymph-nodes and spleen are enlarged, as pseudoleukemias.

You see that we have entered a very involved field, but one of the most interesting in medicine if one will but master a few fundamentals. With the information I have given you, you will be able to orient yourselves in a great number of the cases you encounter. Careful examinations of the blood will enable you to separate from the symptom-complex called morbus maculosus

cases which are without a doubt acute leukemias, pernicious anemias, or septicemias. Nevertheless, in spite of the employment of every available diagnostic method, it will not infrequently happen that we are unable to distinguish between these different conditions *intra vitam*, and a very thorough histologic examination of the organs is essential to a positive diagnosis. This is not the place to discuss the pathologic anatomy and

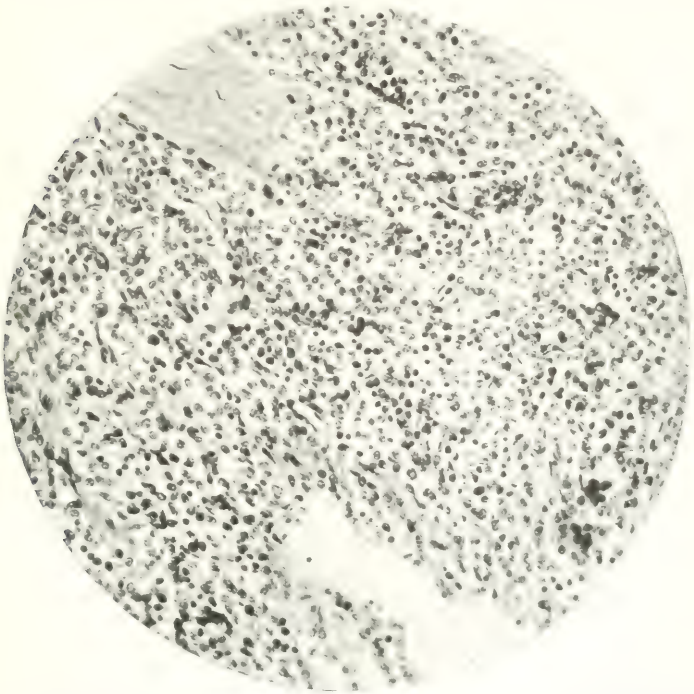


Fig. 130.—Section of spleen from an anatomically verified case of acute lymphatic leukemia, showing the large-celled lymphoid infiltration. (Magnification × 230.)

histology of pernicious anemia, sepsis, and purpura hæmorrhagica. In acute lymphatic leukemia, which concerns us now, the characteristic and determining histologic finding is the lymphoid infiltration of the organs, the cell in the majority of cases being a large one. However, just as in the blood, the lymphatic infiltration in typical cases of acute lymphoid leukemia may be predominatingly small celled.

In the specimen under this microscope (Fig. 130) you will see such a large-celled lymphoid infiltration. The tissue is a section of spleen from a case of acute lymphatic leukemia.

You may ask, finally, why this is a case of acute lymphatic rather than acute myeloid leukemia, as the clinical pictures of the two are practically identical, and as in both conditions there are found these large mononuclear cells with an undifferentiated cytoplasm, in the one case lymphoblasts and in the other myeloblasts. There are various methods known for distinguishing these two cell types. I have already given you some of the morphologic details of the pathologic lymphocyte (page 856). In the case before us there is little room for doubt. Were this a myeloid leukemia we should unquestionably find myelocytes in greater or less abundance as evidence of the transition from the undifferentiated mother-cell to the ripe cell of this system, namely, the polynuclear leukocyte. As you see from the table of differential counts, however, practically all of the cells present except the polynuclear neutrophils are lymphocytes. In other words, the myeloid system is relatively inactive.

This is a case, therefore, of acute lymphatic leukemia, showing practically no involvement of the lymph-nodes and only a slight enlargement of the spleen, with a blood-picture greatly resembling that of pernicious anemia, and belonging, if not strictly interpreted, to the symptom-complex called "purpura hæmorrhagica" or "morbus maculosus."

A final word now as to the therapy employed in this case. The prognosis in acute leukemia, as you are well aware, is unqualifiedly bad, though Türk has reported several cases in children exhibiting typical clinical and hematologic pictures which have recovered. The treatment is purely symptomatic. In our patient, as the bleeding from the gums is the most striking and alarming feature, the chief effort has been to control this.

Repeated injections of normal horse serum exerted little or no effect. For example, I note here a record that on the day following the injection of the serum the patient expectorated 8 ounces of blood in a single afternoon. Transfusion was, therefore, resorted to. The donor was a man with broken cardiac compensation re-

quiring venesection. After preliminary agglutination and hemolysis tests 23 ounces were taken from the donor and injected into our patient. The so-called citrate method was employed to prevent clotting, enough 2 per cent. sodium citrate solution being added to the blood to make a final strength of $\frac{2}{10}$ per cent.

The technic of this operation is very simple: A favorable vein of the donor is dissected out, a cannula or needle introduced, and the blood allowed to flow via a connecting rubber tube to a graduated receptacle containing 2 per cent. sodium citrate solution in measured amount. Constant stirring of the mixture with a glass rod is an additional means of preventing clotting. When sufficient blood has been obtained the amount is noted, and enough citrate solution is added to make its concentration for the whole mixture $\frac{2}{10}$ per cent. The citrated blood is then conveyed by the gravity method from the ordinary normal salt transfusion jar through rubber tubing and cannula to the vein of the recipient.

Referring once more to the record, I find that before the first transfusion the patient was taking absolutely no nourishment, could not sleep, complained of headache, and was markedly dyspneic. Soon after receiving the blood his headache disappeared completely, the breathing became much easier, and the lips showed a light pink color. The following morning the bleeding from the gums had ceased entirely, the mucosa being covered by a closely adherent dark coagulum, the breathing was normal, and the patient said that he had obtained the first sleep he had had for several nights.

This improvement continued for two or three days, the patient gaining in strength, eating and sleeping well, and the gums resuming almost a normal appearance. Then the bleeding reappeared, however, and his condition again became grave, necessitating a second transfusion. The latter was successful as before in stopping the hemorrhage and in alleviating the other symptoms, but in a few days things were as bad as before. To date he has been transfused on six different occasions, with no permanent results. So far as the patient's blood itself is concerned, transfusion accomplished little that was recognizable aside from producing a moderate increase in the number of red corpuscles.

Twelve hours after the first operation the erythrocytes had jumped from 600,000 to 1,400,000, and thirty-six hours after the third transfusion they had mounted from 1,420,000 to 2,100,000. The clotting time remained at about three minutes, however, and the leukocytes, as already noted, showed a progressive decline.

Today his condition is very grave, and although we are still resorting to transfusion, it is accomplishing less and less, and exitus cannot be far off.

NOTE.—Death occurred three days after the patient was presented in clinic. An autopsy was not obtained.

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